ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

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CONTENTS

I.

A SYNDROME OF EAR AND SINUS SYMPTOMS DEPENDENT UPON DISTURBED FUNCTION OF THE TEMPOROMANDIBULAR JOINT.*

JAMES B. COSTEN, M. D.,

ST. LOUIS.

The problem of temporomandibular joint function and occlusion being a major dental issue, it appears almost entirely in the dental literature. The following group of symptoms may be observed frequently in patients with endentulous mouths and a marked overbite; the syndrome is classic for lesions of the sinuses or ears; yet overbite and disturbance of the joint are so easily overlooked as etiologic factors that it becomes a source of error in analyzing cases in otolaryngology.

Conditions that have been given most attention in the medical literature are anterior dislocations, fracture of the neck of the mandible and ankylosis of the joint after chronic irritation or infection. These have received prolific comment, and appropriate surgical treatment has been carefully worked out.

Each of these symptoms may be ascribed to some evident disturbance in anatomic function of the joint, its ligaments and

muscular attachments. The ear symptoms depend upon actual involvement of the eustachian tube and tympanic structures. The "sinus" symptoms are more apparent than real. The actual source of this group of complaints was confirmed by the marked improvement which followed correction of the overbite, renewal of molar support to take pressure off the condyle, and establishment of proper articulation of the condyle within the fossa.

The ear symptoms observed were:

Impaired hearing, continuously, or with intervals of improvement.

Stopping, or "stuffy" sensation in ears, marked about meal time.

Tinnitus, usually "low buzz" in type; less often, a snapping noise while chewing.

Pain, dull type, within and about ears.

Dizziness, mild; again, attacks of prostrating severity, definitely relieved by inflation of eustachian tubes.

Alleged "sinus" symptoms:

Headache, severe and constant, localized to vertex and occiput, and behind the ears—typical site of posterior sinus pain, but increasing toward the end of day (atypical sinus history, and suggestive of eye headache).

Burning sensation in throat, tongue and side of nose.

The diagnosis of this condition is established by:

1. The lack of molar teeth, or badly fitting dental plates, permitting overbite;

2. Mild catarrhal deafness, improved at once by inflation of eustachian tubes;

3. Dizzy spells, relieved by inflation of tubes;

4. Tenderness to palpation of mandibular joints;

5. Marked comfort to patient from interposing a flat object between the jaws.

6. Presence of the typical headache after sinus or eye involvement has been corrected; presence of the typical headache when sinuses or eyes are found to be negative.

Eleven cases in which disturbance of mandibular joint function was the chief etiologic factor of abnormal ear and head conditions:

Articular eminence

Articular disc

Section through
condyle

Posterior portion of
capsule

Spheno-mandibular &

Stylo-mandibular ligts
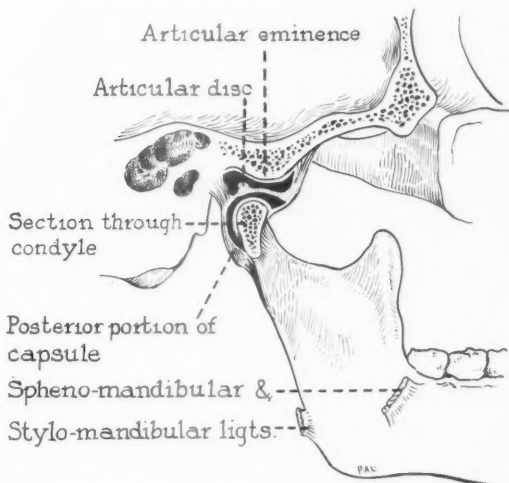
Fig. 1. The temporo-mandibular articulation. Schematic drawing to show position assumed by articular structures when full molar support is afforded (mesial aspect). No pressure is exerted on the meniscus by the condyle.

Temporalis

Pterygoideus int.

Masseter
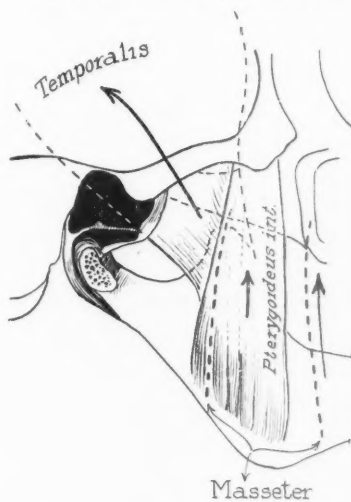
Fig. 2. Variety of movements increased in the pathological temporo-mandibular joint. With molar support gone, condyle is brought upward against meniscus, forward against articular eminence, or backward against tympanic plate.

Fig. 3. Sketch of section through mandibular joint in a cadaver specimen. It demonstrated the effect of uncontrolled movement of the condyle in the edentulous mouth. The bone has undergone pressure (?) atrophy, excavating the glenoid cavity.

Case 1.—Mrs. P. F. N., 61. The past history of this patient was unimportant, the only events being concerned with the ears. The hearing was gradually impaired for a few years, with tinnitus of low buzzing type. The relatives noted her to be more deaf, or inattentive, at meal time; an hour later she seemed responsive and heard much better, denying stoutly any hearing difficulty. The object of examination was to prove whether or not she could hear, and consult a neurologist if necessary. No headache, or respiratory infection of importance. Occasional dizzy spell, attributed to "biliousness." Dental plates 20 years old.

Examination, September 27, 1927: The patient has a good structural nose with no evidence of chronic infection about sinuses or pharynx. Tonsils are small, atrophic. The ear drums are perfect, somewhat dull, slightly retracted. Inflation of eustachian tubes show the left patent, admitting air with normal sound. The right tube is tightly adherent, admitting no air.

Hearing distance is 30 feet on left, WV and CV. On the right, CV is heard at one foot, WV not at all. Weber to right. Diminished positive Rinne, both sides. Bone conduction slightly prolonged over examiners. C1 and C4 reduced on right. Marked overbite of the lower jaw, folding the lower lip over the upper.

Diagnosis: O. M. C. C., right; overbite with eustachian tube compression.

Proper dental plates were advised. No treatment of the ears was possible, as the patient lived in a small town at some distance.

On October 25, 1927, one month after opening of the bite with well-fitted plates, she returned for inspection. CV was increased from one foot to fifteen, and WV from 0 to ten feet. There were no further dizzy spells.

Case 2.—Mrs. A. K., 62. For many years this patient has had a "raw" feeling in the right side of the throat. She has frequent headache, daily, over the right eye, behind the right ear, and into occiput. There is a burning sensation, at the end of the day, in the right side of the nose and tongue, which extends at times to the right ear. No deafness or dizzy spells.

Examination, July 24, 1928: The nasal spaces are clear of infection. The lower turbinates are enlarged, but not occluding. In the nasopharynx, the right eustachian tube is set forward by a smooth mass, mucosa not broken on surface, and about 0.5 cm. in diameter. Palpation shows it soft in texture. The pharynx is otherwise normal. Upper and lower molars absent on right.

Hearing tests show normal hearing, bone conduction slightly prolonged. Biopsy of the mass was suggested and refused.

Diagnosis: Sphenoiditis, chronic, right; pharyngitis, chronic, secondary; nasopharyngeal tumor, right (?).

October 2, 1933. The patient returned after five years' absence. Biopsy was made and sections reported as chronic inflammation, lymphadenoid tissue. Palpation of the mass showed it still soft, not increased in size.

All molars, upper and lower, missing on the right. The right temporomandibular joint is quite tender to firm pressure, and bite closes with slipping of jaw to left, and wrenching of right joint. Observed through nasopharyngoscope, the soft mass in the nasopharynx is seen to bulge markedly on closure of bite.

1. Art eminence temporal bone
2. Coronoid process of mandible
3. Ext pterygoid plate of sphenoid bone
4. Int pterygoid plate of sphenoid bone
5. Cartilage of torus tubarius
6. M pterygoideus ext - upper head
   (spheno meniscus m)
7. Internal maxillary artery
8. Ligament M temporalis
9. Tendon m tensor veli palitini
10. M masseter
11. Auriculo-temporal nerve
12. Inf alveolar nerve

Fig. 4. This section was made in a cadaver specimen with mouth open, and the condyle well forward on the articular eminence of the temporal bone. In this position the sphenomeniscus muscle is taut. When the jaw is brought upward, as in a marked overbite, this muscle and associated structures are seen to relax, and the bulging tissues compress the membranous part of the eustachian tube.

The patient was sent to her dentist, and upper and lower denture fitted in the molar spaces on the right. The last report from her was three weeks after, and there was marked relief from the headache and burning sensation.

Case 3.—Mrs. W. H. B., 33. Patient is just beginning her allergic reaction to the fall grasses. In addition to the usual symptoms of sneezing, watery discharge, and obstruction of the nose, she has had a regular dull headache, localized about the ears and radiating to the cheek bones. The pain is daily and becomes severe by the end of the day. A burning sensation radiates to the right ear. Onset corresponds with extraction of lower molar teeth six months before.

Examination, July 30, 1932: Nasal spaces show allergic reaction, throat negative. Tonsils removed. Much dental work. Lower molars missing on

both sides, and the lower incisors close behind the uppers with an extreme overbite.

Diagnosis: Headache from mandible joint overaction; overbite of jaws —extreme.

On May 20, 1933, patient returned with previous headache symptoms more severe. No trace of her seasonal allergy may be seen in the nose. Complains of "full feeling" in right ear; examination negative—tube normal. She was again urged to have proper fitting of jaw teeth, and this time did so.

On July 2, 1933, about one month later, she reported that all of her headache had disappeared. It did not return with her hay fever in the late summer.

Case 4.—Mrs. B. B., 63. For past two years there has been a noticeable impairment for whispered voice, with middle and low tones heard well. No tinnitus. No severe illness, or quinine medication. No headache or dizzy spells. All upper teeth have been extracted and an upper plate worn about nine years.

Examination, August 15, 1932: The nasal spaces are of fair structure, except for a low spur forward on the left. Ear drums perfect. Tonsils removed, small fragment in the left lower pole. The upper teeth are replaced by a plate, lower teeth in good repair. There is a forward overbite of almost one-half inch, and the upper incisor teeth edges cover the lower gums.

Hearing distance, CV is thirty feet, both ears. WV is heard at one foot on left, not at all on right. Weber not lateralized. Rinne normal. Schwabach, bone conduction time approximates that of examiners for normal, 40 seconds.

On June 19, 1933, patient reported, after wearing new upper plate for six months. Whispered voice increased to five feet, both ears. There was great difficulty in making a proper plate, and occlusion is still obviously very bad.

Case 5.—Mrs. J. S. M., 55. This remarkable case has been an invalid with paralysis agitans symptoms for four years. The tremor was confined to the hands, weakness and gait difficulty prevented any effort at walking, and attacks of mild dizziness made the patient refuse to sit up in bed. The hearing was impaired, with sensation of "stopped ears." There was a dull temporal and occipital headache, daily. The presence of occasional infection about the posterior sinuses seemed to prove this the source of the headache, and in view of the tendency to become worse, a resection of the posterior sinuses was considered. The gravity of this procedure brought in consulting advice.

Examination, November 15, 1932: The nasal spaces were entirely normal except for some hyperplasia about the posterior sinuses. The pharynx was negative. Ear drums perfect, slightly dull and retracted. Very flat, and poorly fitting plates, 22 years old, permitted an extensive overbite of the jaw. Palpation of the mandibular joint showed marked tenderness, both sides.

Hearing distance for spoken voice was thirty feet, both ears, whispered voice not heard. C4 and C gross forks not heard, middle C heard well. Weber not lateralized. Bone conduction prolonged to sixty seconds. Rinne

Spheno-
mandibular
ligaments

A

1 ----

2 ----   A'

B

Broken lines   spheno-mandibular ligaments
and pterygoid muscles further relaxed by marked
overclosure of edentulous mouth

Jaw open, condyle forward, spheno-mandibular
ligaments tense (Prentiss)

Mandible closed, condyle resting in glenoid
fossa - relaxation of spheno-mandibular
ligts incorporated in the fascias of the two ptery-
goid muscles (Prentiss)

A'- Coronal section through ramus and
the two pterygoids to show sphenomandibular
ligament a part of general fascia (1)Ext ptery-
goid (2)Int pterygoid (Prentiss)

Fig. 5. Sketch of a study by Prentiss, showing the manner of tensing the spheno-mandibular ligaments, and their behavior in a closed bite. Movement of the pterygoid muscles is uniform with the ligaments, being controlled by them.

positive. Eustachian tubes were tightly stopped when inflation was attempted.

New dental plates were fitted, and the eustachian tubes inflated weekly for one month. The dizzy spells disappeared at once, the headache was completely relieved by the end of the month's period. This report is one year later, at which time the paralysis agitans symptoms have gradually increased. The patient sits up in bed or wheel chair without dizziness; there is no further headache; the hearing is improved.

Case 6.—Mrs. L. W., 66. The patient notices impairment of hearing, which is gradually worse, and is more marked at meal times. No tinnitus or "stopped" sensation of ears. In spite of good general condition, she has had dizzy spells, requiring support during an attack. Dental plates 15 years old.

Examination, November 29, 1932: There is a good structural nose, with a small spur on the septum. No infection in any sinus group. Drums are dull, somewhat retracted. Fork tests show all tones heard well except C gross, which is lost in both ears. No change from normal bone conduction, Rinne, or Schwabach. Weber to the right.

Diagnosis: O. M. C. C. bilateral. Dental plates fitted loosely, and in addition to a wide overbite, allow the jaw to slip over one side in occlusion. New plates were advised, but patient could not buy them. She refused to obtain them at a charity clinic.

June 15, 1933, while having a dizzy spell, the patient fell, fracturing right femur; recovery after seven weeks.

September 3, 1933. On this date she fell, breaking several ribs.

The hearing is improved and the dizziness disappears as long as she can report weekly for inflation of the eustachian tubes. After explaining the condition to her, she has had marked relief, also, from holding a small pad of gauze between the jaw teeth.

Case 7.—Mrs. S. L., 55. This patient was admitted on the neurological service at Barnes Hospital on April 18, 1933. Routine examination was entirely negative. She had a history of headache, covering a period of ten years. The pain was dull, distributed over eyes, in the parietal region, and around the ears. It had become more severe the past winter. No hearing impairment or ear symptoms. She was referred for sinus study.

Examination, April 19, 1933: The sinus groups were negative except for a small amount of mucopus in the middle meati. Throat negative, ears normal. X-ray study showed maxillary involvement on both sides, and clouding of the left frontal.

The 18-year-old dental plates showed a wide overbite, with a ludicrous weaving of the lower jaw during attempted occlusion. Tenderness may be elicited on palpation of each mandibular joint.

She was advised to have the mouth refitted with proper dentures, before attempt to correct the rather inactive sinus condition. This was promptly done. She reported by letter after four months, that all of her headache symptoms had entirely gone.

Case 8.—Mrs. O. J. K., 64: For several years this patient has had a pain or ache which is occipital, worse on the right side, and during the daily attack has a low buzz in the ears. No history of nasal infection. Family notice hearing impairment, but she is not aware of it.

Examination, April 30, 1933: Shows good structural nose, no trace of infection observed. The pharynx is normal, tonsils small. Ear drums are dull, slightly retracted. The lower plate, which is ill fitting (10 years old), allows a marked overbite. Two tongue depressors held flat between the jaw teeth is a noticeable comfort to the patient. (The patient then remarked that holding the mouth open at night always prevented headache. She reasoned that this kept the "cold air" out of her nose and helped the headache.)

Fork tests were within normal range, except for hearing C4 and C gross poorly. Hearing distance normal for CV, reduced to 10 feet for WV.

Diagnosis: O. M. C. C., mild; overbite with pathological joints.

On May 13, 1933, patient reports wearing a new lower plate one week, with complete relief of her symptoms, and improvement in hearing.

On August 1, 1933, a further report from the patient, that she has had no further symptoms, and hearing is improved.

Case 9.—Mr. J. W. T., 47. The patient complains of recurrent attacks of "stuffiness" in ears for several years, with muffling of sounds. He has been treated by an otologist for one year with transient improvement. During the year he had a nasal operation—resection of right sphenoid. He still has an occasional occipital headache. The hearing involvement corresponds roughly with the extraction of all his molar teeth for arthritis five years ago.

Examination, June 2, 1933: The ear drums are normal except for slight retraction. Nasal spaces show sphenoid resection, right side, thin mucopus pouring from all posterior sinus groups. Pharynx is negative. There is
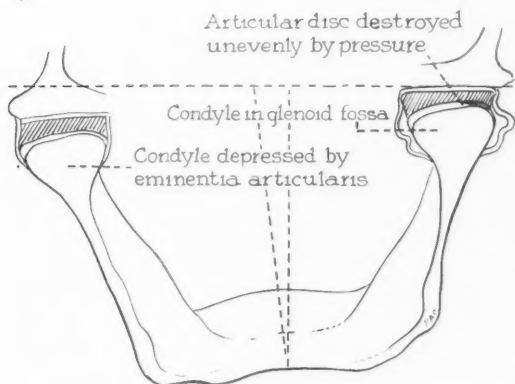
Fig. 6. Asymmetric position of condyles due to uneven support of molars or lack of support on one side. (Prentiss.)
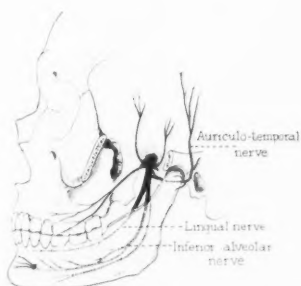
Fig. 7. Sketch showing the distribution of the mandibular nerve and the course of the auriculo-temporal nerve. (Henle.)
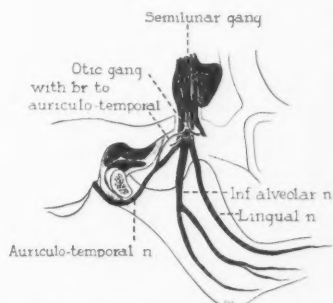
Fig. 8. Diagram of mesial view of mandibular nerve showing auriculo-temporal nerve and its connections with the otic ganglion. (After Spalteholz).

marked malocclusion of the remaining teeth, and, with all molars extracted, the lower incisors rest on the hard palate, one-half inch behind the upper.

Fork tests within normal limits. Hearing distance reduced for WV to ten feet on right, and for SV to six feet. Both are normal at thirty feet on left. The distance is improved by inflation of the right tube to fifteen feet, both SV and WV.

On October 5, 1933, after wearing inlays of molar teeth three months, hearing tests show the same improvement as obtained on the first inflation. He has had no further "stuffy" sensation in ears, and no further headache. The dentist has had extreme difficulty because of his badly deformed jaw.

Case 10.—Mrs. C. F. G., 55. Fifteen years ago this patient had a severe influenza, with nasal trouble ever since. She definitely associated the nasal infections with loss of her upper teeth. Any severe cold seemed to localize in one sinus or other. All teeth were extracted six years ago. This attack dates from a severe cold one month before examination, with daily supraorbital headache, and profuse nasal discharge.

Examination, September 19, 1933: Generally the nose is somewhat crowded, but of good structure. Heavy mucopus in both middle meati. Ears normal. All teeth are extracted, and on first examination it was not noted that there was any abnormal jaw position.

The antra were irrigated several times, with prompt improvement in nasal infection. In spite of this, her headache persisted, and when she returned in one month X-ray study was made, and operative treatment of sinuses considered. Before proceeding, however, she was sent to a prosthetic dentist, who reported retrusion of the condyles on chewing, due to improperly fitted plates. These were changed, and all remaining headache symptoms improved. Sinus operation is still advised, in view of her serious handicap of chronic sinus infection.

Case 11.—Mr. E. A. F., 73. This patient has a remarkably negative past history, and is still in perfect health. He recently had a thorough routine physical examination because of dizzy spells, which were increasing in frequency and severity. All findings being essentially negative, he was referred for the question of toxic labyrinthitis.

He stated that the attacks were brief, severe enough only to occasionally reach for support. There has been some stopping of ears, and impaired hearing. Tinnitus, a low crackle, only when he yawns widely to open ears.

Examination, November 15, 1933: The nasal spaces are free of infection, structures good except for broad septal spur on the left. Pharynx negative. Ear drums normal.

Fork tests show reduction for C4 and C gross, hearing distance reduced to twenty feet for both CV and WV, both ears. No spontaneous nystagmus.

Diagnosis: O. M. C. C., bilateral, mild.

There is a marked overbite of his badly fitting plates, the jaw instantly feeling more comfortable when resting the molar teeth on three tongue depressors, equal to about one cm. in thickness.

Inflation of the eustachian tubes entirely relieved the dizzy attacks and he proceeded to replace the old plates. All his symptoms are improved with the new jaw position effective a few days.

Wright[1] and later Decker[2] reported cases demonstrating compression of the cartilaginous canal by habitual retrusion of the condyles of the mandible. These authors ascribe deafness in those individuals to: (1) Compression of external canals to point of closure, (2) trauma to tympanitic structures and irritation from the continued click and pound as the condyles slip backward with each closure of the mouth.

Zygomatic
    process

                                    Eustachian tube

Mandibular
      fossa
Petrotympanic
      fissure

                                    PA Conrath

Mastoid
      process
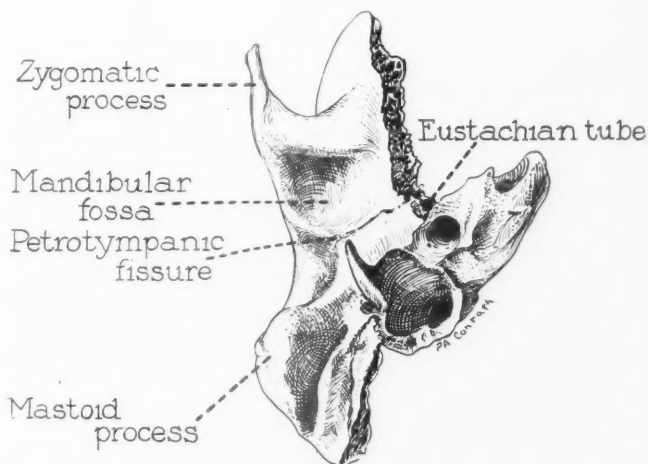
Fig. 9. Lower aspect of temporal bone, showing the petrotympanic fissure
quite high, within the depression of the mandibular fossa. In such relation
as this, the chorda tympani nerve emerging at the mesial end of the petro-
tympanic fissure, is subject to irritation from the movements of the condyle
in a pathological mandibular joint. (Drawn from a specimen in the
anatomical collection of Dr. R. J. Terry, Washington University School of
Medicine.)

If these deductions are accepted, it would be on the basis of
some type of concussion of labyrinthine structure or injury to the
eighth nerve, in which case the recovery of hearing after correct-
ing the joint function would be very slow. Their cases, as well
as most of the present series, promptly improved in hearing within
a few days or weeks after restoration of proper occlusion. The
temporary nature of such deafness seems clear. The symptoms
of dizziness in cases 1, 5, 6, 8 and 11 temporarily disappeared
with the first inflation of the eustachian tubes and hearing distance
was improved during the test. The rôle of compression of the
eustachian tubes and a resultant conduction deafness was quite
evident; and the association of dizziness with improper regulation
of the intratympanic pressure was repeatedly demonstrated. Cases
5, 6 and 11 habitually practiced opening the mouth as in a yawn
to relieve dizziness and coincident "stopped" feeling in the ears.

Looking to anatomic reasons for such a pressure effect, we find
a definite basis for compression of the tubes. A section (Fig. 4)

made through the articular eminence close to the glenoid fossa.
passes through the attachment of the sphenomeniscus muscle to
the articular disc, and through the lumen of the eustachian tube;
it embraces all soft structures adjacent to the tube. With the
joint in normal position, the external pterygoid muscle is taut
(Fig. 5-A), and the tensor veli palatini muscle borders the tube
anteriorly on an almost straight line (Fig. 4-9). Between these lie
only connective and adipose tissue, and posteriorly, close to the
tube, are the auriculotemporal nerve and the inferior alveolar
nerve. If the jaw is brought upward into a position of marked
overbite, the upper head of the external pterygoid muscle (called
the sphenomeniscus muscle by Prentiss[4]) is relaxed and a bundle
of soft tissue piles against the tube. (Fig. 4.) The tensor veli
palatini muscle appears loose, preventing its function in tighten-
ing the soft palate and opening the eustachian tube during deglu-
tition. Especially during the act of swallowing, when the tensor
palatini muscle usually opens the eustachian tubes, the compress-
ing effect of the tissues on the tube from the overbite is present
and prevents it.

The very looseness of the capsule of the mandibular joint and
its restraining ligaments now works to further exaggerate the
pushing of tissues toward the tube. (Figs. 5-B.) With each over-
acting closure of the mandible by the masseters the internal ptery-
goid and temporal muscles, the condyle is shoved upward against
or through the atrophic or perforated meniscus, or it moves back-
ward to the tympanic plate and pushes mesially on one side or
the other through the loose capsule. This happens countless times
with each meal, when the patient retrudes the jaw to occlude his
poorly fitting plates.

Prentiss[3] observed with wide variations of thinning of the
meniscus that perforations were produced on various areas. Its
location would depend upon the angle at which the condyle was
forced against the meniscus; such an uneven pressure follows the
unilateral loss of molar support. (Fig. 6.)

The overbite with atrophy of the joint structure occurs at once
if the dental plates are poorly fitted and allow it. It develops
slowly if the same plates are worn for many years, and shrinkage
of the bone in the dental ridges is marked.

Goodfriend[5] recently gave exhaustive reports on the symptom-atology and treatment of abnormalities of mandibular articula-tion, with emphasis on the mechanics of repositioning the mandi-ble and establishing the proper dental occlusion. His analysis of ninety-one cases shows only 12 per cent of the group are aware of the joint symptoms and that the majority of them seek treat-ment for the associated reflex symptoms. Deafness leads the list of primary complaints, and then come, in order of frequency, bite anomalies, speech defects, snapping of joints, tinnitus and eczema of ear canals, facial deformities, pyorrhea, malocclusions, and lastly, vertigo. Audiometric examinations were made in an otolaryngologic clinic and these showed 13.3 per cent loss of hear-ing for the abnormal group, but the type of deafness was not reported.

This important investigation demonstrates the following points:

(a) That the patient mentions deafness most frequently as a primary complaint, vertigo last, and headache not at all. (This means that the patient does not associate the two symptoms, and that the vertigo is mild and recurrent over a long onset period. In my group, cases 6 and 11 were referred by internists suspecting toxic labyrinthitis.)

(b) That treatment is sought by the majority of patients for headache and referred pains, the result rather than the unsuspect-ed mandibular joint disease.

The anatomic explanation of pain in connection with disturbed joint function is fairly simple, if recognized: (1) deep erosion of the bone (Fig. 3) of the glenoid cavity leaves only a thin plate between the condyles and the dura—practically nil (Prentiss[3]). Each closure of the jaw impacts this evacuated area with the con-dyle, which thus rocks in the glenoid fossa, barely separated by the remaining thin bone from the dura and its rich nerve supply. (2) With some of the chewing movements and closures of the jaw, the condyle exerts pressure on or near the auriculotemporal nerve which passes intimate to the mesial side of the capsule and between the condyle and the tympanic plate to distribute over the temporal region. (Fig. 7.) (3) Further, in the type of patho-logic joint in which the condyle snaps backward over the articular disc, impacting and eroding the tympanic plate, the chorda tym-

pani nerve passes this spot through the iter chordæ anterius at the medial end of the Glasserian (petrotympanic) fissure. (Fig. 9.) It is therefore quite evident that dull vertex pain from this source may be of dural origin, that pain over the temporal region originates in irritation of the auriculotemporal nerve, and that the pains referred to the side of the tongue may be attributed to pressure on the chorda tympani nerve.

SUMMARY.

Headache and ear symptoms directly dependent upon disturbed function of the mandibular joint frequently occur in cases showing sufficient pathology about the sinuses to otherwise account for them. There are so many medical, rhinologic and ophthalmologic reasons for headache distributed about the ears, vertex and occiput; and there are so many nasal changes to account for eustachian tube obstruction, that evulsion of the condyle of the mandible from overbite is not considered.

Hearing tests show a mild type of catarrhal otitis with eustachian tube involvement, usually simple obstruction. This is due to pressure on its anterior membranous wall, transmitted through soft tissue from the relaxation of pterygoid muscles and associated sphenomandibular ligaments during overbite.

The promptness with which the ears improve seems to controvert the idea that the ear condition is due to trauma or concussion of the labyrinth or tympanic structures from the condyle of the mandible. Cases of shock to the labyrinth from a blow on the chin are not within the scope of this paper.

Attacks of dizziness in these cases are obviously due to changes in intratympanic pressure affecting the labyrinth. The effect is transient and recurrent, relieved by inflation of the eustachian tube, and not the picture seen in toxic labyrinthitis.

The areas involved in the headache cases are typical of headache of posterior sinus origin and are easily taken for such. Persistence of the headache after indicated sinus surgery is sometimes due to mandibular joint pathology.

The symptoms arise as a result of overaction of the joint at first, and later adds the regional effect of a loose, pathologic joint,

produced by absorption of the meniscus, condyles and surrounding bone.

The prognosis in a given case depends on these factors: (a) the accuracy with which refitted dentures relieve abnormal pressure on the joint; (b) the extent of injury to the tube and to the condyle, the meniscus, and the joint capsule.

The mechanics of occlusion and dental problems are not included here. Only sufficient reference to the anatomy of the mandible and joint is made to clarify the ear or sinus diagnosis.

Anatomic reasons are advanced to account for abnormal conditions of the eustachian tube, and for the distribution of pain toward the vertex, occiput, pharynx and tongue. It is barely possible that mandibular joint pathology may be an etiologic factor in glossopharyngeal neuralgia, the association of chorda tympani and auriculotemporal nerves with the ninth occurring via sensory connections to the otic ganglion.

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BIBLIOGRAPHY.

1. Wright, W. H.: Deafness as Influenced by Malposition of the Jaws. Journal of the National Dental Assoc., Dec., 1920, p. 979-92.

2. Decker, J. C.: Traumatic Deafness as a Result of Retrusion of the Condyles of the Mandible. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 34:519-27, 1925.

3. Prentiss, H. J.: A Preliminary Report Upon the Temporomandibular Articulation in the Human Type. Dental Cosmos, 60:505-14, 1918.

4. Prentiss, H. J.: Regional Anatomy, Emphasizing Mandibular Movements With Specific Reference to Full Denture Construction. Jour. of Am. Dental Assoc., 10:1085-99, 1923.

5. Goodfriend, D. J.: Symptomatology and Treatment of Abnormalities of the Mandibular Articulation. Dental Cosmos, 75:844-52, 1933.

Ibid. 75:947-57, 1933.

Ibid. 75:1106-11, 1933.

II.

DIFFUSE CRANIAL OSTEOMYELITIS COMPLICATING FRONTAL SINUSITIS.*

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Diffuse cranial osteomyelitis is the most frequent serious complication of frontal sinusitis. Mackenzie,[1] in 1922, collected forty-four cases of this disease, forty-one of which originated in the frontal sinus, and three in the maxillary antrum. Of this number, twenty of the patients developed an osteomyelitis of the skull as a sequel to operations upon the frontal sinus, and all of them died. Of twenty-one who developed the osteomyelitis as a complication without previous operation upon the frontal sinus, fourteen died and seven recovered (mortality rate of 66.6 per cent). With such unfavorable results it is quite obvious that we have not yet reached a satisfactory solution of the management of this disease.

Furstenberg,[2] in a recent discussion of osteomyelitis of the skull, says that he was unable to find more than seventy-three cases reported, but that this number represents a very small amount of the total number that have occurred. From such a limited observation of so treacherous a disease, it is not difficult to understand why we have not yet settled upon a definite and more satisfactory method of treatment.

Hastings[3] concluded that spontaneous healing of the inflammation of the bony walls of the frontal sinus is hindered by meddlesome surgical interference, particularly when the periosteum, which is the real mesentery that furnishes the bone its nutrition, is stripped. He compromises a bit, however, by suggesting an

opening in the floor of the frontal sinus during an acute inflammatory exacerbation of a chronic condition, saying that this offers surgical relief and gives nature a chance.

Lynch[1] was one of the first to announce the importance of leaving the periosteum undisturbed on the anterior wall of the frontal sinus. No doubt many patients have died with a complicating osteomyelitis, meningitis or brain abscess on account of the failure of the surgeon to appreciate the importance of not disturbing the nutrition to the infected bone. Such surgical procedures are a distinct handicap to the natural defenses in the presence of an acute infection.

My experience with this disease is limited to eleven cases, which I shall report in detail, and from these observations I have drawn the following conclusions:

First, that conservative treatment offers the greatest hope for recovery. By conservative treatment I mean a minimum amount of surgical interference. Since it is an accepted fact that surgical trauma to the acutely infected frontal sinus frequently produces osteomyelitis of the cranial vault, then it is only logical to assume that similar procedures on acutely infected bone elsewhere in the cranial vault would serve to spread the disease.

Second, that even when the disease is recognized early, and where every opportunity is offered for surgical relief, operative interference should be delayed until the acute bone infection has subsided.

Third, that surgical intervention on the acutely infected bone in no way controls the spread of the disease, but, on the contrary, is more apt to cause it to spread.

Fourth, surgical interference during this acute stage disturbs the normal blood supply to the affected part and inhibits nature's effort to control the disease.

CASE REPORTS.

Case 1.—I was called to see M. R., a 15-year-old boy, August 29, 1931, on account of a painful right eye, severe and constant headache, which was progressively becoming worse, fever of 104°, and drowsiness. His history revealed that five days before admission he went swimming in an outdoor pool. He dived several times and did considerable swimming

under the water. The following day, as he was stooping over playing
croquet, he had a sudden severe pain over his right eye. Within a few
hours the upper lid began to swell, and he was quite tender over this area.
The head pain was all limited to the forehead. It became progressively
worse, he was unable to sleep that night, and the following day he had
a hypodermic injection of morphine for relief. He became nauseated and
vomited once. As the pain increased, so did the swelling and tenderness
about the right eye. He became drowsy, and although his fever was 102°,
the pulse rate was 64. I saw him six days from the onset, and, with this
previous history and an X-ray picture that showed distinct clouding of the
right frontal sinus, I decided that the patient probably had a brain abscess,
as an extension from a flare-up of an old frontal suppurative sinusitis.

Under ether anesthesia, I made an incision through the right brow and
found the right frontal sinus filled with thick yellow pus. The posterior
wall was removed and an old pachymeningitis was exposed. This area
was covered with thick yellow droplets of pus. The history of the devel-
opment seemed too rapid for a brain abscess, but I felt certain that I
would find a collection of pus directly beneath the old infected dura. I
was disappointed in this conclusion because incision through this area and
exploration into the frontal lobe of the brain revealed no pus. This ex-
ploration was not very extensive, however, as I felt that the history was
too brief to warrant an extensive surgical exploration at this time for a
brain abscess.

The following day he was no better, and was brought by ambulance
one hundred and fifty miles to Kansas City. He was quite exhausted from
the trip; semi-stuporous, continued with a slow pulse, and a temperature
of 103°. The next day he had a hard, generalized convulsion, after which
he was completely paralyzed in his left leg and arm. These convulsions
got progressively worse. His drowsiness increased, and I decided to ex-
plore his right frontal and right temporal regions for a brain abscess. No
abscess was found in the right frontal region on deep exploration.

The dura was exposed over the right rolandic fissure, but, on account
of the extensive pachymeningitis and osteomyelitis in this region, which
was still acute, it was deemed inadvisable to go through the dura at this
time. Also, the patient was in a very bad condition for further surgical
exploration. He was returned to bed, rallied from the operation, and,
within forty-eight hours, there was a recurrence of the convulsions. They
were almost continuous, and ether anaesthesia was required to control
them. These convulsions started in the lower left leg, and came up the
left side of the body, involved the left arm, the left face, and the head
was always turned to the right. Finally, one ampule of 50 cc of 50 per
cent glucose was given intravenously, and the convulsions promptly
stopped.

The following morning the patient was much better, was rational, alert,
ate a good breakfast, and I noticed that thick yellow pus was draining
from beneath the dressings overlying the exposed, but unopened, dura
about the rolandic fissure. Much to my surprise, when the dressings were
removed, I found that pus was oozing freely through a small dural fistula
in this area. This opening was not disturbed, but continued to drain very
freely, and the patient rapidly improved. Within three days he was using

Fig. 1. Appearance of patient three weeks after draining brain abscess following osteomyelitis. The abscess drained through the right temporal incision.

his left arm and leg, and within three weeks' time he was out of bed and walking about the ward. The supraorbital wound rapidly healed (Fig. 1) and small sequestra of bone worked out through this incision for several weeks. The patient has been well since without any complaints.

Comment: Brain abscess as a complication of osteomyelitis of the skull is not uncommon, but a spontaneous drainage of a brain abscess, where only the overlying bone has been removed and the dura not even incised, is very unusual. Unfortunately, I explored very thoroughly the frontal lobe of the brain where there was no abscess, and did not explore the area of brain where the abscess was located, even after I had exposed the dura. My reluctance, however, seems to have been justified, since the patient made such a prompt and unexpected recovery. Also, the slow drainage is in line with the proper treatment of brain abscess. I am convinced that this abscess was subdural, and was secondary to the osteomyelitis and pachymeningitis. It is interesting to note also that the patient has recovered from the osteomyelitis with only a minimum amount of bone drainage, and without any attempt to remove all of the infected bone.

Case 2.—Miss M. T., aged 21 years, was admitted to Isolation Hospital March 3, 1924, with measles. She had been ill about one week, and had a diffuse, straw-colored, irritating discharge from both nares. Two weeks later a swelling which had developed about the inner angle of the left eye, was incised and drained pus. This was apparently coming from an

infection within the left frontal sinus, which had broken through the external plate. This continued to drain for about ten days, when the wick was removed, and she was apparently making satisfactory progress. She was up and about the hospital and visited from the window with friends outside. Suddenly, thirty days after her hospital admission, she became unconscious, had a slow, shallow respiration, and a pulse rate of 50. A diagnosis of brain abscess of the left frontal lobe, secondary to an osteomyelitis of the frontal bone, was made.

At operation the brain was explored through the left frontal sinus and a large subdural brain abscess, containing about one and one-half ounces of pus was drained from the left frontal lobe. The mucous membrane lining of the frontal sinus had been completely destroyed. There was a large sequestrum of the outer table and a smaller one of the inner table. This latter sequestrum was so firmly adherent to the dura that, in removing it, the dura was torn and the abscess opened. Pure growths of staphylococcus albus were cultured from the bone chips, sequestrae and brain abscess.

Comment: This patient has made a satisfactory recovery and has been normal in every way since her operation. She later told me that she had had trouble with her left frontal sinus as long as she could remember, and suffered excruciating periodic attacks of left frontal headaches. This, I am certain, was the background for the development of her brain abscess. On examining her nose I found a sharp deflection of the septum to the left, which was later corrected by resection.

While it is hardly advisable to await the development of a brain abscess before surgically interfering in osteomyelitis, yet this patient did manage to survive both complications and illustrates that, if left alone, osteomyelitis of the frontal bone may localize.

Earlier surgical interference might have prevented the complication of the brain abscess, yet, without her measles, it might not have occurred. She might also have succumbed to the osteomyelitis if operated upon too early.

Case 3.—F. B., a 10-year-old girl, entered St. Luke's Hospital April 2, 1929. Two years prior to admission the patient went swimming while suffering from a cold, and developed severe frontal sinusitis. Following a few local treatments, she apparently was well. Two weeks prior to entrance her mother noticed a swelling on the left side of her forehead. She had no pain for two years and no headache. She had no complaints except the swelling on her forehead, which had been tender in this area since her first sinusitis.

Physical examination showed a postcervical adenopathy and an elevation on the left upper forehead, about the size of a hazel nut. Examina-

Fig. 2. The dark area above the midline of the left orbit is an old area of osteomyelitis with liquifaction of the inner and outer table and small sequestrum floating on a bed of dural granulations.

tion otherwise was negative. The laboratory report showed a hemoglobin of 80 per cent; R. B. C., 4,200,000; leucocyte count, 10,400; urinalysis, negative; blood Wassermann, negative.

The patient was operated upon April 7, 1929, under ether anesthesia. An elliptical incision extending over the brow from the midline, one-half inch below the hair line, was made. The periosteum was intact, and, when elevated, light yellow pus oozed forth from a subperiosteal abscess. The outer table of bone was necrosed. A sequestrum, a half centimeter in diameter (Fig. 2), as shown by X-ray, was floating in a mass of old broken down granulations which had as their base, the dura. The bone edges of this crater were removed with the gouge, the adjoining area was uncovered, and the bridge work of bone lying between the two was removed. This necrosed area did not erode the outer table, but the inner table was completely gone, and the dura was covered with the same type of granulations. The rough bone borders were cleaned off, the wound was sutured, and one strip of gauze wick was inserted. The frontal sinus and intracranial structures were not disturbed. The wound healed by primary intention (Fig. 3). The gauze wick drain was removed on the third day, and the child left the hospital six days following her admission. The convalescence was uneventful, and at no time since her operation has she had any trouble.

Fig. 3. Photograph taken one year after the removal of a seques-
trum of a dormant osteomyelitic process involving the left frontal
bone. The incision was made near the hair line in order to better
obscure the scar, which having healed by primary intention without
drainage, is very inconspicuous.

Comment: A series of X-ray pictures taken over a period of
two years following her primary nasal infection, reveals the
progress of her localized osteomyelitis of the frontal bone. Early
in the course of her disease I recognized the fact that she was
developing an osteomyelitis secondary to her frontal sinusitis.
Due to the high mortality rate of this disease, and to the fact
that the patient was having so little discomfort, I was reluctant
to disturb the lesion. Also, as previously mentioned, it struck me
as bad surgery to attack this infected bone during the acute stage
of the involvement. I also felt that this frontal sinusitis might
clear up spontaneously and independently of the osteomyelitis.
Especially, since this latter was well removed from the frontal
sinus.

Here again was a very similar condition to the infected mas-
toid process. Also, if I had gone into the acute and infected
frontal sinus, I believe that this chronic infection of this frontal
bone, which had already broken down and become walled off,
might have been reinfected and a re-exacerbation of the osteomye-
litis would probably have taken place.

It is also interesting to note that the organism—i. e., the staphylococcus albus in pure growths—was cultured from the bone chips, the sequestrum and the epidural abscess.

Her uneventful convalescence was due, I believe, to the time interval between the onset and the operation (two years), which allowed thei nfection to become walled off, lowered the virulence of the organism, and changed an acute and dangerous virulent infection to a mild chronic bone abscess.

Case 4. Mrs. M. H., age 57, entered St. Luke's Hospital June 17. 1926. on account of swollen right eye of five days' duration. She had a temperature of 99°, 6800 leucocytes, a negative Wassermann and a normal urine. The patient stated that she had had a "sinus headache" and constant head colds since an attack of influenza eight years before, and that two weeks prior to admission she had taken cold and had been unable to sleep without sedatives on account of a severe frontal headache and pain above and behind her right eye.

Examination showed a marked swelling and redness of the right upper lid, which extended up over the eyebrow and down over the right cheek to the angle of the mouth. The right frontal sinus and right antrum of Highmore were extremely tender. There was a moderate amount of thick, purulent discharge coming from the right middle nares. The intranasal mucous membranes of the right nares were engorged.

The following day, under ether anesthesia, an incision was made through the right upper brow, and thick, yellow pus was encountered in the superficial soft tissues. The periosteum was detached from the outer table, and loose sequestrae of bone were floating in the purulent debris which entirely filled the frontal sinus. The mucous membrane lining the sinus had been destroyed, and when this debris was removed, a small area of dura, covered with dense granulations, was found near the posterior inferior angle. The sequestrum of bone over this area had completely sloughed out, and the dura adjacent to the bony margin was firmly adherent to the inner table. The bony margins of this opening were not disturbed, and no attempt was made to remove the old epidural granulations. The external wound was sutured, and a cigarette drain was left in the inner angle for five days.

Cultures from this pus and bony fragments showed a pure growth of staphylococcus.

The patient made an uninterrupted recovery, and four years later she states that she has had no trouble with her head, and has been free from colds and headaches. An X-ray taken at this time shows there has been no recurrence of the osteomyelitis.

Comment: When this patient reached me she had already passed through the acute stage of her osteomyelitis of the frontal bone. A severe frontal headache, which she had long complained of, was apparently due to the dural irritation which had taken place dur-

ing the formation of the sequestrum of the inner table. Complete freedom of head pain since her operation is rather conclusive proof of the above statement. The sequestrum of the inner table, no doubt, antedated that of the outer table, and the abscess in the superficial tissues coming late in the course of the disease, brought the patient to the hospital for surgical relief at a most appropriate time. The findings in this case, and her postoperative course, again illustrate the advantage of later surgical interference in osteomyelitis of the frontal bone.

Case 5.—Mr. R. C., age 55, entered St. Luke's Hospital August 8, 1929. For nine years he had had a profuse discharge from his nose, following an attack of what must have been suppurative frontal sinusitis. Most of the discharge came through the left nostril. It was yellow, thick, and always relieved the pain he had over his forehead. About five years ago he noticed a small mass appearing in his right inner canthus. This followed an inflammatory reaction about this area. At no time had he any blepharitis. This mass gradually enlarged so that it pushed his right eye outward. Otherwise, his vision was not disturbed.

At the time of entrance to the hospital he had a mass about half as large as a hen's egg, which was firm, smooth and not tender nor inflamed, fixed at the base, which fluctuated, and, on deep pressure, yellow, thick, foul-smelling pus could be expressed and drained through the nostril.

An elliptical incision, extending through the right brow and down over the left side of the nasal bridge, disclosed an abscess surrounded by a dense pyogenic capsule. The under surface of the right frontal sinus was entirely necrosed and connected directly with this abscess. The frontal sinus, bony septum, as well as the frontal ethmoid septum and the bony partitions of these sinuses, were completely destroyed. Also the mucous membrane lining of these sinuses had sloughed out, leaving smooth, bare bone. This opening into the frontal sinus was enlarged to about one inch in diameter, until a normal appearing bone was reached. The naso-frontal opening was enlarged, an iodoform drain was inserted through into the right nostril up into the frontal sinus. The pyogenic capsule was dissected out and the orbital incision was tightly closed. No dura was exposed, and the orbital capsule was not opened into.

Comment: Due to the long standing infection and the loss of virulence of the organism, I felt in the beginning that this patient should make a complete recovery if the nasofrontal drainage could be maintained. About three years ago he had a stab incision into this abscess with drainage, but of course without relief. Intranasal surgery alone might have taken care of this condition, but would probably not have led to complete healing on account of the bone necrosis of the outer table. There was profuse nasal

discharge, but the patient made an uneventful recovery, with wound healing by primary intention.

Case 6.—B. P., age 36, was first seen in 1926. He came to me on account of the swelling over the inner canthus of the left eye, which pushed the eye outward and disturbed the vision. There was a continuous purulent discharge from the left eye and the left ear. This trouble dated from a severe attack of scarlet fever at eleven, at which time had a septicemia, pneumonia and acute multiple arthritis. He was ill in bed three months. The tip of his right mastoid process sloughed out at this time. He had been unable to breathe through the left nostril since this illness, and had a profuse, foul-smelling discharge, which bubbled out of the left inner canthus when air was forcibly expelled through the nostril. The left nares was almost completely closed by adhesions connecting the superior and middle turbinate with the septum. An X-ray revealed a loss of the bone tissue about the inner angle of the left orbital plate extending into the frontal sinus.

Under a local anesthetic, by the intranasal route, this encapsulated abscess of the inner canthus was drained, and sequestrae of bone from the floor of the left frontal sinus was removed.

The patient made an uninterrupted recovery. The eye returned to its normal position, and he had no nasal discharge afterward. There has been very little improvement in hearing, but the suppuration from the middle ear has stopped.

Comment: Twenty-five years' duration of this bone infection had certainly afforded time enough for the patient to develop a compete immunity. It was unnecessary, however, for him to endure the suffering over such a long period of time. When infection does attack the bony processes of the skull, following scarlet fever, it frequently produces the most extensive type of destruction, with the tendency to become chronic, as is so well shown by the persistent discharge from the middle ear.

Case 7.—Mrs. M. S., following an incision of an upper lip furuncle, was admitted to the surgical service of the Kansas City General Hospital on July 22, 1927, suffering from a painful, swollen, infected upper lip and left side of face.

Three days prior she had consulted her family physician on account of a small sore on her left upper lip. The doctor incised this red, tender area, and got a little pus by squeezing vigorously. This was very painful, and, promptly following this trauma, she developed a high fever, the whole left side of her head became intensely tender, swollen and painful, and the eye swelled shut.

Twenty-one days following the onset, I saw her in consultation. She was so exhausted she was hardly able to answer a few questions. Following the extreme scalp and face tenderness multiple fistulas had formed on the entire left face and were draining freely a yellow, thick, purulent discharge. The entire tissue resembled the surface of a broken down abscess,

and, in fact, the entire left scalp and upper face was undermined. Pressure upon this area caused pus to spurt from the fistulae without pain, and over the left scalp deep fissures in the outer table of the skull could be palpated. While the left eyelids were still matted together with pus, there was good vision and no proptosis or chemosis. The wound of the upper lid had healed, leaving a triangular scar. The cellulitis did not involve the neck or the occiput and the ears were normal.

On August 14, four days later, the patient died without developing any symptoms of meningitis, and apparently expired from exhaustion due to the widespread virulent infection. The X-ray of her skull showed a diffuse, spreading osteomyelitis. No autopsy was permitted.

Comment: The interesting part of her progress was not that she developed such a fatal complication following the trauma to the upper lip, but that the widespread thrombophlebitis did not involve the cavernous sinus. Instead of taking the usual route via the angular and the ophthalmic veins, direct to the cavernous sinus, the infection turned off via the superficial temporal and involved the diploetic veins of the skull.

Mackenzie,[1] who reports an interesting case of spreading osteomyelitis of the skull, recommends vigorous and early surgical interference. However, I doubt that this patient was ever a proper surgical subject for such a radical operation, as it would have been necessary to do a hemicraniotomy to remove all the involved bone.

Case 8.—On July 20, 1930, J. C.* entered Bell Memorial Hospital on account of a swelling over his left eye. He had been struck on the back of his head seven days prior by a golf ball. Later, he had been in swimming. There was marked chemosis and proptosis of his left eye, and, at first, it looked as if he were developing a cavernous sinus thrombophlebitis. The opposite eye, however, was not involved, and the orbital swelling extended upward on the forehead.

A diagnosis of a supraorbital abscess was made, and deep incision into the supraorbital fat drained off a considerable quantity of light yellow pus. An incision on the forehead also broke into an abscess, which was subcutaneous, but not subperiosteal. This latter abscess was apparently a direct extension from the orbital abscess upward over the soft tissue. Following this drainage, the child made no improvement. His tempera-

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*Two years later this boy was re-operated on account of convulsions, which, over a period of eight months, had increased in frequency and severity. A few subdural adhesions were found. A sequestrum of newly formed bone was removed and the frontal lobe of the brain was explored, but no abscess located. There was an absence of convulsions for a few weeks following this operation, but since that time the convulsions have re-occurred. It is possible that this boy still has a brain abscess.

ture remained high, fluctuated between 101° and 105°. His pulse rate was 80. He became very irrational and required large doses of amytol to keep him quiet. While dressing the orbital wounds he developed a clonic convulsion of his right side, starting in his right face and proceeding to his right arm and leg. When awake, he complained bitterly of occipital pain. The eyegrounds were normal. He apparently had no visual disturbance, and his other physical findings were normal.

The patient continued to have headache, had a marked ankle clonus on the right side, and four days later had a slight convulsion which was limited to the right side. He became drowsy, irritable, with a pulse rate of 66, and a leucocyte count varying from 8,200 to 10,250.

At this time I was firmly convinced that he was developing a brain abscess of his left frontal lobe. An X-ray of his head at this time, however, was suggestive of an osteomyelitis of the left frontal bone, which was positively confirmed by a picture taken on the sixteenth of August (twenty-three days later) which showed destruction of the outer table. Also at this time the patient made marked improvement. He became alert and playful, had no more convulsions, had intermittent attacks of severe headache, vomited some, but was not projectile in type, ran a daily temperature of around 100°, and at this time we noticed a slight paralysis of the right side of his face. The clinical confirmation of the osteomyelitis of the skull was proven by a dome-like swelling over the center of his forehead, which was not red or tender. As the patient's general condition improved, this swelling increased.

After ten days' observation, with continuous improvement, the swelling over his right forehead began to subside, and there was a copious discharge of thick, yellow pus from his left nares. He was up and about the ward, but complained that he developed an "awful headache" if he stayed up too long.

At this time he was transferred from my service, and a radical removal of all the infected bone over the left frontal region was done, even up to the normal bone. From the operative sheet I learned that the adjacent bone was quite soft and easily chipped away with Rongeur forceps. The surgeon states that this bone was chipped away in all directions, even up to and including normal bone as he thought that with this radical procedure it would be the only method of cure for this patient. Several areas of granulation tissue about the dura were exposed, and this granulation tissue was scraped away down to the dura. The sequestrated areas extended from the brow upward for 6 cm, and the width was about 5 cm. The bone overlying the frontal sinus was removed, the inner table was not destroyed. It was very definitely determined that the osteomyelitis had arisen from the left frontal sinus, as an area of exposed dura was found in the posterior border of the left frontal sinus. Iodoform gauze packs were inserted, and the incision was closed in layers.

The patient made an uninterrupted recovery, although the surgeon stated, "The prognosis at this time is not any too good; however, with this radical treatment, the patient is given his best chance." This proved in this particular case to have been a satisfactory method of treatment, yet I still feel that the radical surgery which involved the disturbance of non-infected bone entailed considerable risk, and that the chief factor in

the uneventful recovery was not so much due to the radical surgery, as to the fact that the patient was operated on one month from the onset of the disease.

Comment: In looking over the patient's course, it is not difficult to see why I was so convinced that he had a brain abscess. His course is almost identical with that of case number eleven, yet it is not very much unlike that of case number ten, who did have a brain abscess. This patient's convulsion, headache, photophobia and other symptoms of brain abscess were brought about by the dural irritation which probably extended through the dura and produced a pachymeningitis interna. That the patient was able to manage this most serious part of his complication without the aid of surgery is again a point in favor of early conservatism in the management of this disease.

Case 9.—Luetic osteomyelitis. H. L. B., age 37 years, came to the hospital January 8, 1923, on account of a swelling above both eyes, and a tender, swollen mass on top of his head. Twelve years before he was struck on his right cheek by a baseball, and the cheek became painfully swollen, the swelling lasting for several weeks. Six years later he began to have severe pain over his frontal sinuses, which he described as bone pain. The pain was constant and became worse at night. This lasted for three or four years, during which time he had considerable purulent drainage from his nose. In 1921, his right eye became swollen and the swelling extended up over the right forehead. This was lanced, and drained considerable pus. He was in bed five weeks, and then apparently recovered. Two months prior to entrance to the hospital the pain over his right eye and swelling over both eyes returned. He again had this swollen mass incised, much pus drained off, and he was relieved of the pain.

When I first saw him, on his admittance to the hospital, there was marked edema of the supraorbital region, with a draining fistula just above the right inner canthus. There was a marked nasal depression, and also an irregular depression over both frontal sinuses. There was a swollen mass just above the hair line in the center of his forehead, one-half as large as a dime. This area was not particularly tender, was quite firm, and seemed to be attached to the periosteum. There was a profuse discharge from his nose, with almost complete destruction of the nasal septum. The X-ray pictures of the skull showed all of the nasal sinuses cloudy, and the entire frontal sinus eroded. The patient had a normal temperature, normal blood count, and a negative urine. His physical examination otherwise was negative. The blood Wassermann was four plus, and under anti-luetic treatment he rapidly improved.

Comment: Luetic osteomyelitis of the cranial bones is not extremely uncommon, but may, in the early stages, be mistaken for a non-luetic lesion, as happened in this particular case. Radi-

cal surgery is, of course, contraindicated, particularly at the onset
of the disease. I believe the trauma was the etiologic factor in
this particular case, and the secondary infection was the primary
cause of the extensive bone destruction.

Case 10.—Mrs. H. S., a 34-year-old woman, was brought to St. Luke's
Hospital on July 11, 1931, on account of severe pain in her left upper jaw.
The first and second left upper molars had been extracted just prior to
her admission, and this post-operative pain had become so severe, that
she required repeated hypodermics of morphine. She had a temperature
of 100°, 12,000 leucocytes, normal urine, and her general physical exam-
ination was otherwise negative.

Fig. 4. Frontal view showing a sequestrum and destruction of
the inner and outer table over the right frontal bone.

About four years prior to her admission, she had had some very ex-
tensive radical surgery of all the nasal accessory sinuses. There were
two vertical scars extending upward on the forehead directly above each
inner canthus. The right vertical scar had drained intermittently for three
years following her operation, and the scar tissue had now a very un-
healthy appearance. On palpation over this area, a distinct ledge of bone
could be felt. The X-ray pictures showed a defect in the bone about the
size of a five-cent piece. Another X-ray picture taken directly over the
forehead (Fig. 4) showed a very definite sequestrum formation with de-

struction of the inner and outer table of bone. Her temperature increased, she became nauseated, and complained severely of frontal headache.

While under observation, she began to have chills with sharp elevations of temperature of 104°. There was a very scant excretion of urine which was now loaded with pus cells. She complained of low abdominal pain, had frequent urination with much burning. This bladder disturbance was thought to account for her chills and fever, and after ten days' treatment her condition cleared up, her fever subsided, and the area of frontal osteomyelitis was explored.

A transverse incision, three-fourths of an inch above the brow, was made connecting the two perpendicular scars. The periosteum was firmly adherent over the site of the osteomyelitis on the right side. The bone around this fistula was partly broken down, and the outer and inner table were destroyed. The dura was covered with old, thick granulations, and a loose sequestrum was lifted off. The lesion above the left eye did not involve the inner table. The outer table was softened, bled freely, and the dura was not exposed. A vaseline gauze pack was left undisturbed in this wound for ten days, and within three weeks the entire area was completely healed.

Comment: It is probably fortunate that this lesion had been overlooked for three years. No doubt the osteomyelitis was of traumatic operative origin, and, due to the chronicity of her sinus infection, the invading organism was of low grade. The dural irritation caused by the presence of the infecting foreign body— i. e., the sequestrum—was just enough to produce the head pain. As the wound healed, drainage was impaired, and the symptoms became progressively worse. No attempt was made to remove any bone, except a few sharp edges, and the loose sequestrum. The open drainage permitted healing from the floor of the wound. No attempt was made to connect this area with the frontal sinus, as this procedure, in my opinion, permits the entrance of a new bacterial flora from the nose, and good drainage through the nose is difficult to maintain.

We must bear in mind that we are dealing with an entirely different type of bone infection than osteomyelitis of the long bones, where the medulla is involved. Also, it should be borne in mind that the dura, which forms the inner periosteal covering, is one of the most resistant tissues to infection that we have in the body. If the infection does not extend through the inner wall of this membrane, and produces a diffuse meningitis at the onset of the disease, then we are quite safe in awaiting the subsidence of the acute infection. When diffuse meningitis of the fatal type

does occur coincident with the onset of the osteomyelitis, we may
be assured, and should also derive some consolation from the fact,
that no manner of medical or surgical therapy could have fore-
stalled this tragic complication.

The orthopedists stress this thought in their discussion of
osteomyelitis of the long bones, when they say if the patient sur-
vives the first attack of osteomyelitis, regardless of the secondary
involvement of the other bones, the patient will rarely succumb to
the disease.

In treating osteomyelitis of the skull, I believe that we should
be prepared to make certain sacrifices, and should always bear
in mind the very important fact that we must first save our pa-
tient, and then consider the prevention of complications and the
treatment of the local lesion. It is impossible in the beginning to
predict the extension of the osteomyelitis of the skull, just what
bones will be involved, and just how much intracranial involve-
ment will take place.

The time element should also be carefully evaluated, as we
know that as any disease persists, the virlence of the organism
is lowered and the resistance of the patient to the bacteria is
increased. Nowhere in the body is this better illustrated than
in osteomyelitis of the cranial vault. Soft tissue involvement may,
at the onset, present a very hazardous complication, and it is par-
ticularly dangerous to disturb the soft tissues about the face, when
acutely infected, on account of the probability of the development
of thrombophlebitis, which may extend to the cavernous sinus
or downward to the internal jugular vein.

Case 11.—On the 26th of June, 1931, I was called to Pittsburg, Kansas,
to see a 24-year-old man with the following history:
Ten days before, he went to a dance, became quite warm, and then rode
in an open car about eight miles and went in swimming. The following
day he felt badly, had a headache, and his nose was stopped up. He stayed
in bed, developed a pain over his right eye, but thought that this was due
to an eye strain because he had been reading too much. The pain became
worse; he had no chill, but the following day had fever, and his right eye
and the entire right face were swollen and tender. This swelling extended
down over the right cheek, backward behind the right ear, and along the
angle of the jaw. His upper lip also became swollen, and he was unable
to close his jaw because the inner border of the cheek pressed between
the teeth. There was no evidence of any superficial infection about his

face. He continued to get worse, had a temperature of 102°, developed
a marked chemosis of the right eye and was unable to move the lid.

When I first saw him he was lying quietly in bed, was rational, answered
questions clearly, but was quite drowsy. There was no rigidity of his
neck, his reflexes were normal, and there seemed to be a bulging of his
right eye. His temperature was 103.4°, and he had 24,000 leucocytes.
Due to the history of a cold in his head and swimming, I did not feel
that this patient had a cavernous sinus thrombosis, although his clinical
picture completely fulfilled all of the necessary requirements. Although he
had a marked cellulitis of his entire right face, which was a secondary
involvement, and the swelling about the eye more closely resembled an
orbital abscess.

I made an incision through the right brow, and promptly opened into an
orbital abscess which contained about an ounce of thick, yellow pus (Fig.
5). That night he rested fairly well and his temperature came down to

Fig. 5. Appearance of patient prior to first operation. The swelling
about the right face is suggestive of cavernous sinus thrombosis.
This was due, however, to an acute infection of the soft tissues and
osteomyelitis.

100.4°. The following morning at ten o'clock he had a peculiar spell,
which the nurse thought was a fit, following which his left leg and arm
were completely paralyzed. While talking to him at three o'clock that
afternoon, he had a very slight convulsion which lasted only a few min-
utes. He said that he had no headache, and the swelling about the right
eye had been considerably reduced. He complained of severe pain in his
epigastrium and beneath the sternum. He had a slight cough and ex-
pectorated bright red blood. On extreme effort, he was able to move his
left arm and leg.

The swelling on the right cheek looked to be a pterygoid abscess, and
five days later considerable pus was drained through an external incision.
The temperature subsided, he took fluids freely, he had no more pain in

his chest, no cough and no bloody sputum. The eye swelling continued to increase.

Within a week he left the hospital, but three weeks from the onset of his trouble he developed a severe frontal headache. These pains would come suddenly without warning, and last only a few minutes. They occasionally awakened him from a sound sleep. At no time had he been nauseated or vomited. His bowels moved regularly, he took plenty of fluids, and, when free from pains, had a ravenous appetite.

On account of the persistent head pain he was brought to St. Luke's Hospital, where an X-ray of his skull revealed the cause of the disturbance; namely, an acute osteomyelitis of the entire right frontal bone. The primary focus had apparently originally been in the right frontal sinus. Instead of developing a cavernous sinus thrombosis, he had developed this extensive bone lesion. The patient was kept under observation for a few days, and developed a soft swelling which extended obliquely from the center of the top of the skull to the outer angle of the right brow.

Under local anesthesia, an incision was made through the scalp at the right angle of the brow, and considerable pus was drained off. The head pains were diminished, the fever subsided, and the paralysis of his left arm and leg had so improved that he was up and about the ward. During this semi-convalescent stage, while the patient was visiting in the city, he suddenly had a hard convulsion, became unconscious, and was brought back to the hospital.

I saw him the following day and he did not remember anything of his experience. Except for the pain over the right side of the head, which had increased, he was no different than the day prior to his convulsion. I was convinced, however, that there was a dural irritation over the motor area of his brain, and elected to give him better drainage.

Under local anesthesia, the scalp was incised directly over the swollen area extending from a mid-point at the top of his skull to the draining fistula at the right angle of his brow. An old pyogenic capsule, made up of dark red granulation tissue, was uncovered, and this inflammatory mass in several areas was lying directly upon the exposed dura. The inner table of the skull had been completely destroyed in various areas, and numerous loose sequestrae of bone were lying in the granular debris. The wound was left wide open and loosely packed with vaseline gauze. There was rather profuse drainage, and for three weeks loose sequestrae of bone worked out. It was several weeks before the wound entirely healed, but during this time the patient had no head pain, no convulsions and no fever.

The X-ray had already shown an extension of the osteomyelitis involving the superior and inferior orbital plate, as well as the right zygomatic arch. At times the eye had been swollen shut, and small sequestrae of bone had worked out through the draining fistula of the upper lid. This had been accompanied by more or less pain. Due to the patient's continued improvement, and absence of any signs of intracranial complications, as well as the extensive involvement of the bone about the orbit, I had elected not to disturb this area, but had preferred to permit these sequestrae to make their spontaneous exit (Fig. 6).

Fig. 6. Incision extending down to the dura made under local an-
esthesia and left open to permit better drainage. Sequestra of bone
worked out through this incision.

Comment: My first impression of this patient was that he had
a cavernous sinus thrombosis, and that his condition was inopera-
ble. The duration of the proptosis, with limitation to one eye
with no evidence of meningeal involvement, practically ruled out
this diagnosis. As cavernous sinus thrombosis is always fatal, the
exploratory operation seemed justifiable. Had the pterygoid fossa
been explored during the acute inflammation, I believe that a
fatal complication would probably have resulted.

As we have no means of combating the spread of osteomyelitis
of the skull, I believe it is well to accept such a fact and to drain
areas of infection in the soft parts as they localize and point. The
dura here again demonstrates its ability to withstand the most
active infections, so long as they are limited to the external sur-
face. This irritation was so severe, and the pressure upon the
brain was so great, that the patient developed convulsions. I am
inclined to feel that the hemiplegia, which so quickly followed
the exploration of the orbital fossa, was probably embolic in na-
ture and perhaps of arterial origin. Certainly the patient has not

Fig. 7. Appearance of patient one year following operation. There is still a draining fistula beneath right brow and scar formation at outer angle of right eye. Healed fistula through which sequestra of bone exuded. Scalp wound entirely healed.

at any time given any evidence that would point to the positive indication of brain abscess. As osteomyelitis tends to die out, this patient had a good chance for a complete recovery.

It would be hard to conceive of a more extensive acute involvement of osteomyelitis, with apparent recovery (Fig. 7), and certainly nonsurgical measures deserve more credit in the outcome than any surgery which was done on the bone itself. Particular care was exercised not to disturb anything but loose sequestra.

Due to the fact that such prompt and satisfactory results are derived from the drainage of collections of pus in practically every area in the body, the surgeon is often misled into the belief that drainage of an acutely inflamed area will offer similar results. We would not think of attempting to drain a brain abscess until the abscess had completley formed, and even then experience is rapidly teaching us the advantage of delay in surgical interference. It cost the lives of thousands of patients for surgeons to learn the lesson of late drainage in empyema of the chest. The same may be said of such a common and apparently simple complication as peritonsillar abscess.

I believe that it is the surgeon's realization of the possibilities of intracranial complications, and the appreciation of their seriousness, which at times unbalance his better judgment and
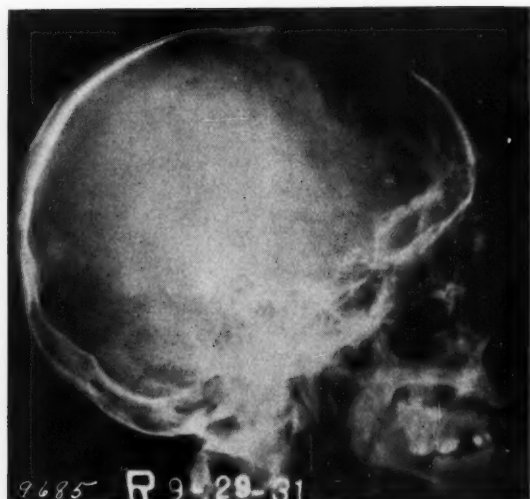
Fig. 8. X-ray taken 102 days from onset of osteomyelitis, showing destruction of outer and inner table of the skull.

prompt him to institute surgical attack upon cranial osteomyelitis which is contrary to all his pathologic reasoning. It is much more difficult to stand by when one has the knowledge of the pathology of this disease and pursue a course of masterful inactivity.

Prolonged convalescence, marked destruction of the cranial vault and repeated minor operations to promote drainage, where it is obviously needed, in no way suffices as an argument in favor of early and radical interference. The scalp is so loosely attached and the outer periosteum is so easily elevated and perforated that considerable and often sufficient temporary drainage will be spontaneously established. This is very well brought out in the postauricular swelling that occurs in mastoiditis, and which is so alarming to the layman and yet so comforting to the patient and consoling to the otologist. Here, nature very beautifully demonstrates her ability to take care of a bone infection and, although recovery may be delayed, there is often a complete cure of the bone infection within the mastoid process by the simple establish-

ment of drainage. This has been so well illustrated by the numerous cures that have occurred after incomplete operative procedures upon the infected mastoid process.

Alarming symptoms and signs indicative of serious intracranial complications may arise either during the onset of the acute stage of the osteomyelitis or at a later time when the patient is apparently making a satisfactory recovery.

Such accidents, particularly when the outcome is fatal, will sorely try the faith of the surgeon in his conservative measures. This is particularly true if he misunderstands the pathology of the complications which have arisen. Furstenberg[2] has stressed the fact that a very important part of osteomyelitis in the pathological development is the inflammation which is set up about the dura beneath the inner cranial table. It is well to remember that the dura is the inner periosteal layer, and that periostitis goes hand in hand with osteomyelitis. Such being the case, it is only natural to expect the patient to develop signs of dural irritation which may closely simulate the picture of a diffuse meningitis. This irritation may be so severe that the patient will have convulsions and hemiplegia to such an extent that the observer will be convinced that the outcome is fatal. The following four pathologic conditions are the chief complications of osteomyelitis of the skull:

First, a localized, even though extensive, pachymeningitis.

Second, a diffuse septic leptomeningitis, which is always fatal.

Third, thrombophlebitis of the intracranial sinuses, which is practically always fatal.

Fourth, brain abscess, which may be subdural or deep within the brain substance.

This last complication, although serious, is by no means always fatal. I believe it is well to mention Furstenberg's[2] original and very valuable observation that the dura, acting as a periosteal layer, encourages the reformation of bone, and he offers this as an argument in favor of the radical removal of all infected bone, even beyond the point of infection. His observation is worthy of consideration, but since it is used in support of the radical surgical removal of the cranium, I believe that it may encourage the somewhat timid and cautious surgeon to over-operate upon his patient, with the thought in mind that the large

bony defect may be partially restored. It also serves to again direct the major portion of our attention to the lesion instead of the patient.

It is my firm conviction that we can, and should, only drain the affected part as the indications arise. Loose sequestræ may be removed at this time purely on the basis of a foreign body. Since we are unable to predict where the lesion will become limited, and what the outcome will be until we learn more about the management of the disease, we must be content to limit our surgical activities with the consolation that if we can do no good, we certainly shall do no harm.

730 PROFESSIONAL BLDG.

BIBLIOGRAPHY.

1. Mackenzie, Dan: Diffuse Osteomyelitis From the Nasal Sinuses Suppurative. Journal of Laryngology, 1913, Volume 28, pp. 6 and 79.

2. Furstenberg, A. C.: Osteomyelitis of the Skull. Transactions of the American Laryng. Rhin. & Oto. Society. 37: January 18, 1931.

3. Hastings, Hill: Osteomyelitis Associated With Frontal Sinusitis. Archives of Otolaryngology. 13:181-86. February, 1931.

4. Lynch, R. C.: Technique of a Radical Frontal Sinus Operation Which Has Given Me the Best Results. The Laryngoscope. St. Louis, January, 1931.

III.

SOME ANNOYANCES IN THE MANAGEMENT OF MALIGNANCIES OF THE ACCESSORY SINUSES.*

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The average practitioner in our specialty is fortunate if he does not see very many cases of malignancy of the sinuses; the diagnosis, course and treatment of such processes have been detailed for us from the vast and carefully studied material of such clinics as those of Hajek, Hirsch, Dan Mackenzie, Holmgren, Beck and New. Nevertheless, one who sees relatively few of these frightful lesions often feels the need of counsel; and it is in such a spirit that these fragmentary observations are presented for your advice and comment. I cannot too highly praise the wisdom and kindliness with which Dr. Otis B. Wight has guided our use of radium in all of this work.

The complex relations of the accessory sinuses, their penetration among and between nerves, vessels and sense organs of vital consequence, renders malignancy therein far more serious, from many standpoints, than that of visceral or external structures concealed by clothing and often permitting complete extirpation. Worries and annoyances to the physician caring for such cases are legion.

Consider for a moment the utter hopelessness of a maxillary sinus bursting with squamous celled carcinoma. Here the spongy alveolar process and granulomatous masses after extraction should have warned the attending dentist a year at least before we saw this woman of 58. Scooping all of the mass possible through the canine fossa, yet finding the cheek infiltrated, we used 1,825

milligram-hours of radium screened only by thin brass. Six ⅓ mc. glass radon seeds were implanted about the facial ulcer five weeks later. Obviously this poor soul was doomed; a vast fungating ulcer destroyed the cheek, after a four months' remission following the radium. She lived in all six months after our discovery of the lesion; a losing fight in which opiates were the final reliance.

The tenacity with which these poor mortals cling to their tattered shreds of life is phenomenal. Faced with blindness, disfigured by leprous deformities, they cover up pain with smiles and their wounds with clean linen. Rarely will they permit members of their families to see their lesions. One of the most courageous and gallant gentlemen I have ever known was a practicing dentist in a neighboring seaport, who discovered failing vision in his left eye. The local rhinologist, Dr. Spaulding, looking for possible sinus disease as an explanation for a paling optic disc and narrowed visual field, found a smooth white mass under the left middle turbinate, which proved to be a massive adenocarcinoma of the entire ethmoid. Stabbing three bare radium needles ladder-like horizontally back under the turbinate into the mass, a total of 630 milligram-hours, unscreened, was given immediately. The reaction within five or six days was severe, with extreme orbital cellulitis and chemosis. Later, almost the entire ethmoid came away as a fibrinoplastic mass, and after three months 465 milligram-hours was similarly administered, carrying the irradiation to the sphenoid, but with much less orbital reaction. Of course the eye was blind; and his insurance companies paid this man total disability benefits, thinking he would be dead within a year. Yet, for such was his courage, he did not complain or tell his wife of his almost certain doom, but went back to his office and to work for over two years longer, until slight loss of vision in the right eye betrayed an invasion of the chiasmal region and sphenoid. Two hundred forty-five milligram-hours of exposure were made through the left sphenoid ostium; the process seemed arrested for another year, when rapid failure of vision betrayed an advance behind the septum into the right sphenoid and ethmoid. One thousand two hundred forty milligram-hours of unscreened exposure crosswise of this growth failed to arrest

it, and death from meningitis followed shortly. The left ethmoid region, exenterated by the earlier irradiation, showed no recurrence.

Blindness and immobilization of an eye by extension behind the globe and into the ethmoid was the lot of a woman whose rare, virulent squamous celled carcinoma of the lacrimal sac, activated by slight plaque dosage of radium over a skin cancer of the nasal bridge, I reported in 1922. At first, owing to successful plastic repair of the lids and complete freedom of the sac region from recurrence, she went about her duties for some years as postmistress of a dusty eastern Oregon village. Seven years later fixation of the globe to the lid and ethmoid was much helped by six ⅓ mc. radon seeds; she went bravely on with the education of her two children for three years longer, perishing at length after the frontal and adjoining cribriform were invaded and permitted meningeal infection. If exenteration of this orbit with massive irradiation had been done five years ago, the process might have been arrested; but her children needed her and she would not give up the time and money necessary.

Another elderly woman in religious orders has lost the lower half of the ascending plate of the frontal on both sides; the frontal sinuses and one superciliary ridge have been obliterated, all by electrosurgery, and a poor, tightly drawn skin still covers her frontal lobes, over which the dura required searing by the spark in the hands of her courageous surgeon, Dr. T. M. Joyce. One eye, ulcerated from exposure by the updrawn lid, required enucleation. Yet, in spite of these ravages, more or less concealed by the headdress of her order, the good Sister's chief concern— *vanitas vanitatum!*—is not with inevitable death, but with the fitting of an artificial eye.

Another type wherein blindness supervened is represented by a youth of 20 whose left tonsillar region, hugely swollen, had been repeatedly lanced "for quinsy" by local practitioners. Pus was never found, and big glandular masses appeared on the side of his neck. Swelling from these structures threatened to force a tracheotomy; since the growth was obviously a lymphosarcoma, intensive deep X-ray therapy was made by Dr. Howarth, with

immediate and startlingly rapid local improvement. After three
weeks in hospital he went home for two weeks, to return in tragic
shape: deaf in the ear and almost blind in the eye of the same
side, the mass was found to be choking the upper nasopharynx,
and death came within another week. Pain was absent, owing to
destruction of the Gasserian ganglion. The soft tissues of the
neck had imposed no barrier to the healing rays, and below the
jaw the swelling had almost disappeared. But up along the caro-
tid sheath, into the ethmoids, sphenoids, chiasm and pituitary and
into the petrous and mastoid, heavy bone had held back the rays
and permitted unrestrained development of the tumor.

Loss of an eye is not so bad if the neighboring malignancy can
thereby be controlled; nor, since prosthetic devices have been per-
fected, should attempts be made to avoid facial deformity by too
sparing sacrifice of skin and muscles. Plastic flaps must not be
constructed too early lest they cover in active islands of cancer.
Better a patch over a clean opening than a recurrence. The
psychologic effect of severe facial deformity is not to be denied,
however; and in spite of the much-vaunted war injury masks
of enameled metal hung from spectacle frames, one is prone to
advise bandages or patches for most persons. Artificial eyes, care-
fully blown out below, are necessary where the antral roof is lost
and the eyelids have been saved. There comes to mind a comely
and courageous woman of 42 who suffered violent pain in an
antrum, long chronic, after rather forcible Lichtwitz lavage by
her local specialist. This antrum, of ten years' standing, had been
stirred up by the extraction of back molars a year or so before,
with loss of some of the antral floor by fracture at that time. An
acute suppurative process had been engrafted upon the old lesion
by the forcible lavage; but our examination, with Dr. Loomis, of
the region of the extraction disclosed spongy, whitish adeno-
carcinomatous tissue. Through the fistula 1,800 milligram-hours
of radium filtered through brass and silver were promptly used;
and in the soft tissues toward the anterior pillar eight 1.5 mc.
gold seeds were implanted. However, since she continued to lose
weight and to suffer severe local pain, a broad exenteration was
made three months later through the canine fossa, disclosing
that from $\frac{1}{2}$ to 1 cm. of adenocarcinomatous tissue lined the roof,

floor and posterior wall of the cavity. Brass-covered needes were used to deliver 1,980 milligram-hours, with severe reaction; the roof, outer and posterior walls and much of the hard palate of that side sequestrated within three months, and the eye was lost following a violent orbital abscess caused by the sequestration. But after two years this huge cavity is clean to the pterygoids, free of cancer, and awaits a soft rubber half-palate for restoration of normal speech. Due to the pterygoid scarring, the motion of the jaw is impeded somewhat, but her brave smile remains.

Facial deformity from seventh nerve paralysis due to basilar invasion is not common; an interesting case of adenocarcinoma of the vault of the nasopharynx complained first of postnasal irritation, later of slight though persistent bleeding, with deep throbbing headache. After biopsy by Dr. Ashley, six 1 mc. gold seeds were planted as high as possible in the growth, behind the right middle turbinate. Before the seeds could be secured, however, fourth nerve paresis was superadded to the facial palsy. After irradiation both the headache and the paralyses improved for a time. With their return, and some paling of the right optic nerve without much loss of vision, deep X-ray therapy was employed for several sittings. Relief, after the initial congestive period, was again marked for many months; but after a year had elapsed the growth had progressed sufficiently to destroy hearing and labyrinthine function in the right ear. Remaining conscious in spite of terrible headaches, she regained motion in the face a day or so before death, which came apparently from respiratory paralysis. Regrettably a postmortem could not be secured where she died. An element of confusion in diagnosis until blood and spinal tests were made arose from her complete alopecia, and a large anterior septal perforation. Suspicion was disarmed, however, by her family of seven fine, healthy children.

Saddest of all, in spite of everything we do, is the mental deterioration which comes from heroic surgical measures involving exposure and herniation of the brain. It is pitiful to realize how these people hope against hope when the crushing truth of serious cancer is told them; but when the very structure of thought is threatened, and we must carry them on with our own courage, it is not always an easy task for the surgeon.

Complaint of burning, tingling and stinging, rarely ever of fullness or of the typical frontal pain of maxillary sinusitis, characterizes many of these individuals. When to this is added slight but persistent bloodiness of an exudate otherwise rather scanty or merely mucoid, careful visual inspection of such affected cavities is indicated.

As an example, a handsome man of 50, whose teeth had all been removed some years before because of pyorrhea, was found to have no retained root fragments and no radiographic shadowing of his antrum. But because of the tingling and sanguinolent discharge, he consented to a canine fossa inspection, at which time the whole posterior wall of the antrum was found soft and crepitant. The outer and inner walls, roof and floor were grossly normal, but examination of the gray-white covering of the posterior wall disclosed adenocarcinoma of high virulence. Two thousand one hundred sixty milligram-hours' exposure of radium screened in brass and silver were made from capsules packed well back. After two months a small hard gland showed up below the angle of the jaw, and three 1.5 mc. gold seeds were planted therein. Slight proptosis of the eye suggested upward extension, but vision remained unaffected; and the antrum, except for rather violent exposure reaction of the hard and soft palate, remained free of growth. He went back to his home for about a month, but returned in frightful condition. The eye was proptosed badly, vision much reduced, cornea insensitive and ulcerated, and he complained of very severe boring headaches. It was explained that his only hope, and that but slight, lay in the utmost radicalism by electrosurgery. Up to this time the patient had been much concerned about his appearance; now, loss of half his face meant little if his pain could be stilled.

Accordingly, Dr. Arthur Jones and I removed the eye, eyelids and cheek, totally exenterated the orbit, took away its floor and the great wing of the sphenoid to the optic foramen, and exposed the pterygomaxillary fossa to the floor of the antrum, fulgurating very carefully because of the proximity of the carotid. An area of the temporosphenoid lobe roughly as broad as the antrum was exposed. Luckily it was not then necessary to tie the carotid, thus

appreciably shortening the time under nitrous oxid. Two weeks later, owing to rather free bleeding from a large branch to the fulgurated pterygoids, the common carotid was clamped under local anesthesia for 30 minutes to be sure that good contralateral circulation existed, then tied, by Dr. E. W. Rockey.

The huge cavity remained fairly clean, but biopsy after four weeks from several widely separated areas still disclosed active cancer cells. Several deep-therapy irradiations were given, especially toward the jugular foramen and the gradually increasing mass of cervical glands, with severe reaction in the palate and throat, but only temporary arrest of the tumor's progress. Potassium permanganate was about the only solution which kept the enormous wound fairly clean. This unfortunate individual, at first meticulous, cleanly and courageous, degenerated into a querulous, sensitive and careless creature of utterly changed disposition, impossible to manage in his home. Back in the hospital, dulled by narcotics, only ten months after he had first noticed the tingling in his upper jaw and two months after the electrosurgical exenteration, he slipped away with a hypostatic pneumonia. On autopsy numerous small nodules of carcinoma were found in the periphery of both lungs; the growth had traveled back over the petrous as far as the tentorium, and was of course highly necrotic where it protruded into the vast facial cavern.

One wonders! Yet, with all these people, what was done lessened their pain, slowed up the inexorable progress of their disease, enabled them to make provision for their loved ones. Perhaps we may recognize such things earlier, by reactions or tests as yet unproven. Perhaps we and our ophthalmologic and dental confreres may watch this field more closely, for earlier signs of danger. If radium or deep therapy are used, it must be expertly managed and dosed to the limit of tolerance. We must be prepared to put up with the annoyances suggested: timidity about radical measures; terror lest relief be too late; fear of visible mutilation; severity of irradiation reactions; danger of sequestration of bone if high irradiation strength be used; foul and unsightly dressings, alarming to other patients; worry over the expense of nursing and hospital care; and, rarely, unreasonable querulousness, to the point of mental deterioration.

Yet not one of these people spoke of suicide, although, save one, they were not religious folk. Bitterly hard as was their lot, they endeavored to spare those dear to them. Complaints were reserved for the doctor's ears. To serve such poor mortals, and in some measure to allay their sufferings, should be reward enough for any physician worthy of his oath.

806 MEDICAL ARTS BLDG.

NOTE ON SOME CHANGES IN THE HYDROGEN ION CONCENTRATION OF NASAL MUCUS.*

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DULUTH.

Some studies of the physiology of the nose and pathology of the common head-cold have been made that required an understanding of the hydrogen ion concentration of nasal secretion.

Consequently a number of both normal and pathologic samples of secretion were tested for their pH values in the ordinary manner, using a series of standardized indicators. The results seem to be worth a brief report.

It was noticed quickly that the pH reading had a tendency to change very soon after the secretion had been expelled. A group of twenty-two samples of normal secretion, a few of which were collected by suction, showed this tendency. The resulting figures varied between 7.2 and 8.3. The more quickly the readings were made the more nearly they approximated the smaller figure. Those samples collected by suction invariably gave higher values than those collected by blowing the nose.

The secretion samples collected from patients with colds showed a similar tendency to change. In order to determine the extent and speed of this change, some single samples were tested repeatedly during the first fifteen to thirty minutes after being expelled. These samples were donated by a patient during the third and fourth days of a cold. There was watery secretion on both days without much obstruction.

The values found in three samples are given in table 1. There was a change from approximately 7.0 to 8.5 in all, during the course of a very few minutes. Thereafter there seemed to be little or no change.

Another sample from another patient was tested on the second, fourth, sixth and eighth days after collection. The values found were, respectively, 8.7, 8.7, 8.6, 8.6.

*From St. Luke's Hospital, Duluth, Minn.

Still other samples were tested two, three, four and five and six days and two and three weeks after collection. All of these gave values in the neighborhood of 8.5. During the interim they were kept in corked vials in the refrigerator.

The cause of this rapid change in pH during the first fifteen minutes, as suggested by Anderson,[1] is without much question due to the loss of CO_2 gas that had been absorbed from the expired air as it passed through the nose. Presumably there is a similar though lesser change in the secretion film within the nose during the respiratory cycle.

In the light of these findings, the small average increase in pH found during colds[2] is probably not significant. It is doubtless due to the decreased aeration of the secretion by expired air charged with CO_2 gas, caused by the nasal obstruction that occurs so commonly during colds.

SPECIMEN 1 EXPELLED AT 6:30 P. M.

Time Tested	pH	Indicator Used
6:30 p. m.	7.4	Phenoid Red
6:31 "	7.6	"
6:33 "	8.0	"
6:34 "	8.27	"
6:35 "	8.27	Thymol Blue
6:40 "	8.30	"
6:46 "	8.4	"

SPECIMEN 2 EXPELLED AT 6:43 P. M.

Time Tested	pH	Indicator Used
6:43 p. m.	7.0	Phenol Red
6:44 "	7.6	"
6:45 "	7.8	"
6:46½ "	7.97	"
6:48 "	8.27	"

SPECIMEN 3 EXPELLED AT 8:08½ A. M.

Time Tested	pH	Indicator Used
8:08½ a. m.	7.17	Phenol Red
8:09½ "	7.39	"
8:10½ "	7.97	"
8:11½ "	8.27	"
8:12½ "	8.27	"
8:13½ "	8.27	"
8:18 "	8.27	Thymol Blue
8:19 "	8.4	"
8:25 "	8.5	"

Table 1.—Changes in pH of nasal secretion found in the first few minutes after being expelled. These three specimens were all collected from a case of head cold on the third day.

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REFERENCES.

1. Anderson, Rubert S.: Personal Communication.
2. Hilding, Anderson: The Common Cold. Arch. of Otolaryngology, XII, 133, 150, 1930.

V.

NERVE FIBERS OF SPINAL AND VAGUS ORIGIN ASSOCIATED WITH THE CEPHALIC SYMPATHETIC NERVES.*

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ST. LOUIS.

The sympathetic nerves in the cephalic region, according to the current teaching, are derived from the superior cervical sympathetic ganglia and consist wholly of postganglionic fibers. These fibers, having their origin in the superior cervical sympathetic ganglia, extend into the cephalic region mainly through the nerves associated with the internal and external carotid arteries.

Clinical data affording evidence of the existence in the head and face of afferent nerve fibers which enter the central nervous system through spinal nerves have long been available. For example, Sluder (1920) described a syndrome having its origin in the orbit or the mucous membranes of the nose and paranasal sinuses which not infrequently includes pain referred to the shoulder, arm, forearm and hand. Herpetic eruptions on the chest accompanying lesions of the nasal mucosa and the mastoid also have been reported. Terracol (1932) cited a case in which operation for the relief of nasal obstruction was followed by eruption on the chest and another in which dressings of the mastoid wound caused violent pains in the arm and eruptions on the chest. Failure of section of the sensory root of the trigeminal nerve to relieve the pain of facial neuralgia in certain atypical cases also has been reported by not a few surgeons. In certain cases the pain of facial neuralgia has persisted following section of the nervus intermedius, in addition to section of the sensory root of the trigeminal nerve. These phenomena have called attention to the possible rôle in atypical facial neuralgia of fibers associated

with the cephalic sympathetic nerves which conduct impulses resulting in pain.

The functional importance of fibers associated with the cephalic sympathetic nerves which may conduct impulses centralwards has been emphasized by the recent work of Helson (1932), who carried out an extensive and meticulous study of the different forms of sensibility in the area of distribution of the trigeminal nerve following section of its sensory root in certain patients and following section both of the sensory root of the trigeminus and the root of the nervus intermedius in others. According to his findings, "the absolute zero of cutaneous sensitivity" in this area cannot be reached by section of the trigeminus and intermedius, but is reached when, in addition to section of these nerves, the cervical sympathetic ganglia also are extirpated. On the contrary, Carmichael and Woollard (1933), in a study of patients with trigeminal neuralgia who had been treated by alcoholic injection of the Gasserian ganglion, patients with facial palsy due to lesions at various levels of the nerve, and patients who had been subjected to injury or extirpation of the cervical sympathetic on one or both sides, obtained no evidence that impulses subserving pain in the face and orbit are conducted centralwards by fibers other than components of the trigeminal nerve.

Foerster, Altenburger and Kroll (1929) published certain clinical data on the basis of which they concluded that afferent impulses arising in the area of distribution of the trigeminal nerve may be conducted centralwards through the cervical sympathetic trunk and upper thoracic nerves. The results of anatomic studies, however, seem to preclude the existence of afferent fibers which traverse the superior cervical ganglion and the cervical sympathetic trunk. Stimulation of the sympathetic trunk between the superior and middle cervical ganglia, furthermore, does not elicit pain (Davis and Pollock, 1932; B. Cannon, 1933).

There remains another pathway along which afferent components of the upper thoracic nerves might extend into the cephalic region, viz., the plexuses on the common, internal and external carotid arteries. On the basis of an analysis of the clinical results of various surgical procedures carried out in the treatment of

atypical facial neuralgia, Fay (1932) concluded not only that afferent components of the upper thoracic nerves extend into the cephalic region along the carotid arteries, but also that afferent fibers of vagus origin join the plexuses on the internal and external carotid arteries.

<div align="center">MATERIALS AND METHODS.</div>

In order to facilitate the anatomic analysis of the nerves associated with the common, internal and external carotid arteries, the following operations were carried out on cats: (1) Unilateral extirpation of the superior cervical sympathetic ganglion; (2) unilateral extirpation of the entire cervical sympathetic trunk; (3) unilateral extirpation of the superior cervical sympathetic ganglion, section of the upper four cervical nerves, ligation and section of the common carotid artery and division of the entire carotid sheath; (4) unilateral extirpation of the entire cervical sympathetic trunk and section of the upper four cervical spinal nerves; (5) unilateral extirpation of the superior cervical sympathetic and nodose ganglia; (6) unilateral extirpation of the superior cervical and nodose ganglia, ligation and section of the common carotid artery and division of the entire carotid sheath; (7) unilateral section of the roots of the upper four or five thoracic spinal nerves just distal to the dorsal root ganglia.

The animals, except those subjected to the last operation, were allowed to live three weeks or longer after operation in order to insure ample time for the degeneration of the interrupted nerve fibers. Segments of the common carotid and the proximal portions of the internal and external carotid arteries with the nerves closely associated with them were prepared for study by the pyridine silver and osmic acid technics. The animals which had been subjected to unilateral section of the roots of the upper thoracic nerves were killed eleven days after operation and the segments of the common, internal and external carotid arteries, with the closely associated nerves, which were removed, were prepared by the Marchi method.

In order to avoid confusion of the carotid sinus nerve with components of the internal carotid nerve, the former was isolated in several animals and prepared for study both by the pyridine-

silver and osmic acid technics. Sections of this nerve prepared
by both technics show that it is composed mainly of myelinated
fibers; consequently, there is little danger of confusing it with
other nerve fiber bundles associated with the internal carotid
artery.

HISTOLOGIC FINDINGS.

Following degeneration of the nerve fibers interrupted by extir-
pation of only the superior cervical sympathetic ganglion, the
nerves closely associated with the upper portion of the common
carotid artery remain practically unaltered. Of the rami along
the proximal portions of the internal and external carotid arteries
some of those which arise directly from the superior cervical
sympathetic ganglion have undergone complete nerve fiber degen-
eration; others contain numerous intact nerve fibers. Certain rami
which are most intimately related to the proximal portions of
these arteries exhibit but little nerve fiber degeneration. The great
majority of the nerve fibers which remain intact in these prepara-
tions are unmyelinated fibers of small caliber. Osmic acid prepa-
rations, however, reveal myelinated fibers in small numbers in
most of the rami with intact fibers, and in large numbers in some
of the rami, particularly in the plexus on the internal carotid
artery.

Extirpation of the entire cervical sympathetic trunk, including
the superior, middle and inferior cervical sympathetic ganglia,
results in more extensive nerve fiber degeneration in the rami
associated with the common, internal and external carotid arteries
than extirpation of the superior cervical sympathetic ganglion
alone, but leaves a goodly number of fibers intact. Extirpation of
the inferior and middle cervical ganglia results in complete nerve
fiber degeneration in the rami which join the common carotid
artery directly from these ganglia. In some cases, sections of the
proximal portion of the common carotid artery, following degener-
ation of the latter rami, exhibit no intact nerve fibers; in others,
they exhibit a few intact fibers, which doubtless are derived from
the plexus on the aorta. Whether any fibers derived from the aortic
plexus reach the internal and external carotid arteries has not
been determined. Sections through the upper segments of the
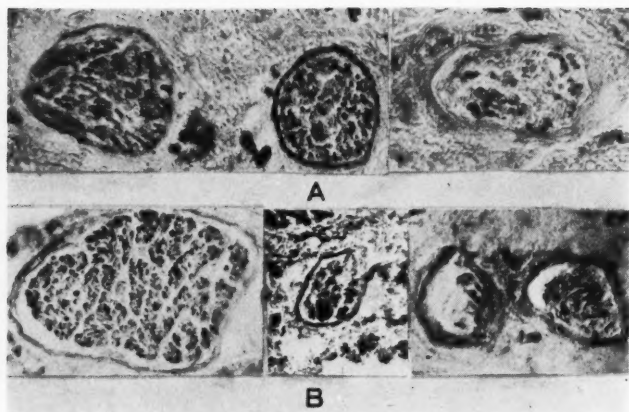
Fig. 1. Sections of bundles of nerve fibers along the internal *(A)* and external *(B)* carotid arteries of a cat three weeks after extirpation of the entire cervical sympathetic trunk, pyridine-silver technic.

common carotid artery exhibit a somewhat larger number of intact fibers, but most of the rami present have undergone almost complete nerve fiber degeneration. The intact fibers in these rami doubtless are mainly of vagus origin.

Sections through the proximal portion of the internal carotid artery exhibit intact nerve fibers in most of the rami present, but such fibers are less abundant in these sections than in sections prepared after nerve fiber degeneration following extirpation only of the superior cervical sympathetic ganglion. Sections through the proximal portion of the external carotid artery exhibit almost complete nerve fiber degeneration in some of the rami associated with this artery, and a goodly number of intact nerve fibers in others. Sections of some of the rami associated with the internal and external carotid arteries, respectively, after nerve fiber degeneration has taken place following extirpation of the cervical sympathetic trunk, are illustrated in Fig. 1. Of the fibers remaining intact in the rami associated with the internal and external carotid arteries, only a small percentage are myelinated.

Section of the upper four or five cervical spinal nerves, in addition to extirpation of the cervical sympathetic trunk, exerts no

appreciable influence on the resulting nerve fiber degeneration in
the nerves associated with the internal and external carotid ar-
teries. Sections through the proximal portions of these arteries,
respectively, after nerve fiber degeneration following extirpation
of the cervical sympathetic trunk and section of the upper four
or five cervical nerves, revealed essentially the same conditions
of the nerves involved in the plexuses on the internal and external
carotid arteries, respectively, as those described above following
extirpation of the cervical sympathetic trunk without section of
the cervical spinal nerves.

Ligation and section of the common carotid artery and division
of the carotid sheath, in addition to extirpation of the superior
cervical sympathetic ganglion, with or without section of the upper
cervical spinal nerves, results in nerve fiber degeneration in the
nerves associated with the internal and external carotid arteries
approximately equal to that resulting from extirpation of the en-
tire cervical sympathetic trunk (Fig. 2). Both pyridine silver
and osmic acid preparations made after nerve fiber degeneration
following this procedure exhibit intact nerve fibers in the nerves
associated with the proximal portions of the internal and external
carotid arteries, respectively, in approximately the same abun-
dance as corresponding sections prepared after extirpation of the
entire cervical sympathetic trunk, leaving the common carotid
artery intact.

Extirpation of the superior cervical sympathetic and nodose
ganglia results in more extensive nerve fiber degeneration in the
nerves associated with the internal and external carotid arteries
than extirpation of the superior cervical sympathetic ganglion
alone, but some of the rami still exhibit numerous intact fibers.
Of the rami present in sections through the proximal portion of
the internal carotid artery, some show almost complete nerve fiber
degeneration; others exhibit numerous intact nerve fibers. The
intact fibers in these rami, however, represent but a small per-
centage of the total number of nerve fibers normally extending
cephalad along the internal carotid artery. None of the rami pres-
ent in sections through the proximal portion of the external caro-
tid artery contain more than a few intact nerve fibers.
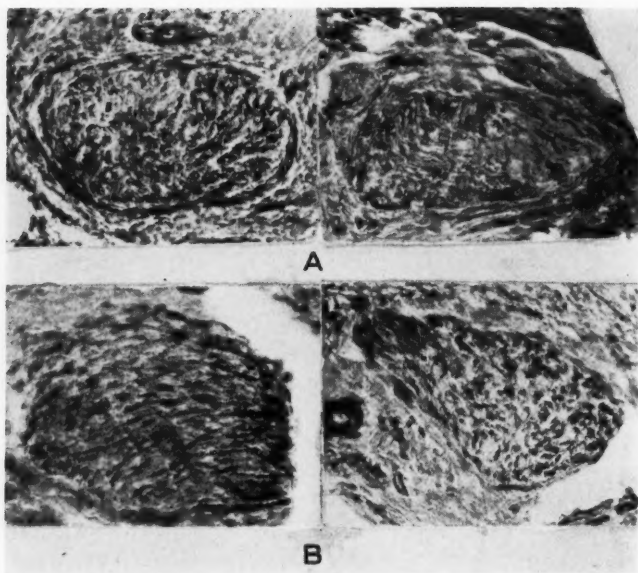
Fig. 2. Sections of bundles of nerve fibers along the internal (A) and external (B) carotid arteries of a cat three weeks after extirpation of the superior cervical sympathetic ganglion, ligation and section of the common carotid artery and division of the carotid sheath and section of the upper 5 cervical nerves, pyridine-silver technic.

Ligation and section of the common carotid artery and division of the carotid sheath, in addition to extirpation of the superior cervical sympathetic and nodose ganglia, results in almost complete nerve fiber degeneration in the nerves extending cephalad along the internal and external carotid arteries (Fig. 3). Most of the rami present in sections of the external carotid artery, in some instances all of them, after nerve fiber degeneration following this operative procedure, are devoid of intact nerve fibers. Degeneration of the fibers in the rami involved in the internal carotid plexus is less complete. In all of our preparations, following this operative procedure, some rami along the internal carotid artery, exclusive of the carotid sinus nerve, still exhibit appreciable numbers of intact fibers.

Section of the roots of the upper four or five thoracic nerves just distal to the spinal ganglia results in degeneration of a goodly
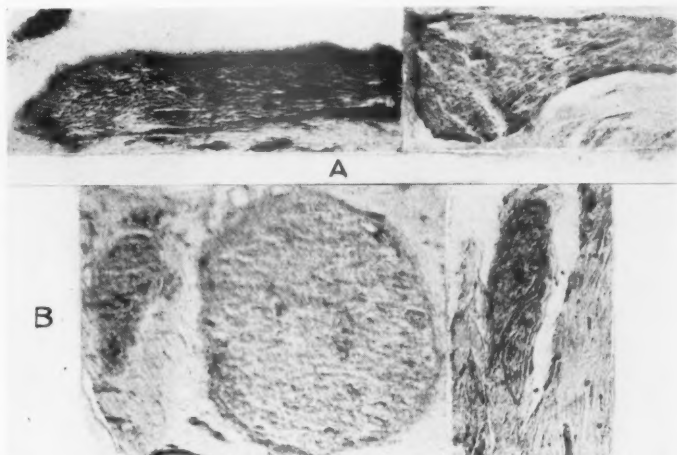
Fig. 3. Sections of bundles of nerve fibers along the internal (A) and external (B) carotid arteries of a cat three weeks after extirpation of the superior cervical sympathetic and nodose ganglia, ligation and section of the common carotid artery and division of the carotid sheath, pyridine-silver technic.

number of fibers extending cephalad along the common and internal carotid arteries and probably a few along the external carotid artery. Sections through the lower and upper portions of the common carotid artery, prepared by the Marchi technic, following this operative procedure, exhibit degeneration of the majority of the myelinated fibers present in one of the rami associated with the artery (Fig. 4, A), but no marked changes in the other rami. The ramus in question also includes many unmyelinated fibers, the condition of which cannot be fully determined in Marchi preparations.

Corresponding sections prepared by the pyridine-silver technic, after allowing a longer time for nerve fiber degeneration, strongly suggest degeneration of some unmyelinated fibers, but the evidence has not been regarded as conclusive. The number of myelinated fibers showing evidence of myelin degeneration in the Marchi sections through the upper portion of the common carotid artery is not appreciably less than in the sections through the lower portion of this artery. Marchi sections through the proximal portion of the internal carotid artery exhibit one or more
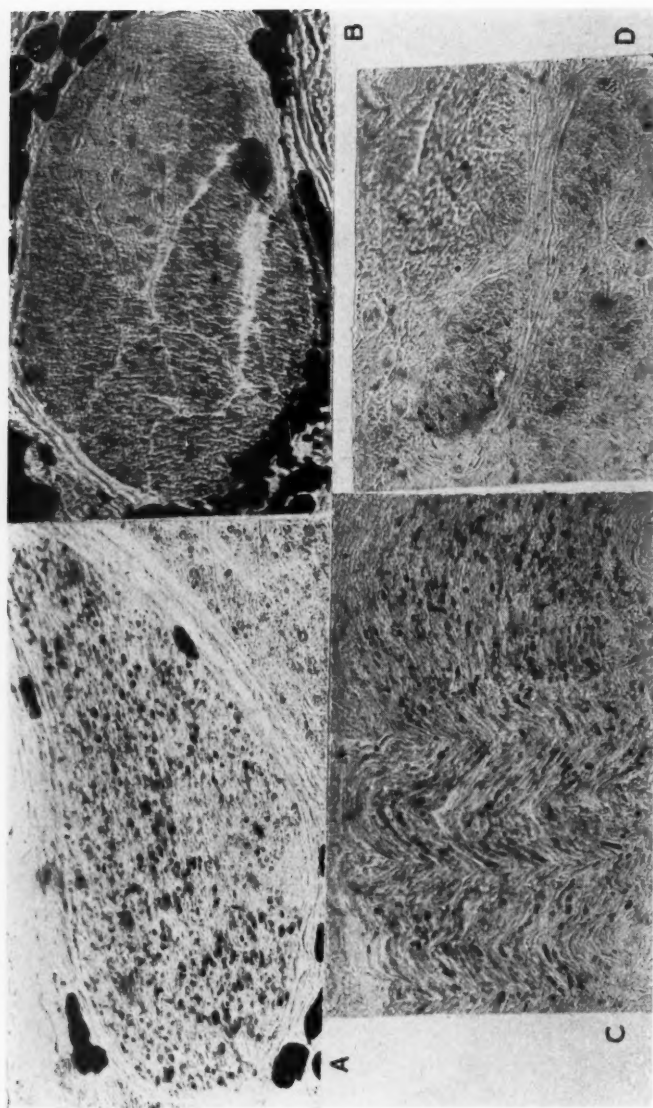
Fig. 4. Sections of nerve fiber bundles along the common and internal carotid arteries of a cat eleven days after unilateral section of the roots of the upper 4 or 5 thoracic nerves just distal to the spinal ganglia. Marchi technic. A, a bundle along the common carotid artery, operated side, showing myelin degeneration; B, the corresponding bundle on the unoperated side; C, a bundle along the internal carotid artery, operated side, showing myelin degeneration; D, corresponding bundles on the unoperated side. The bundle illustrated in C was cut obliquely. The corresponding fibers on the opposite side, as illustrated in D, constitute several bundles.

bundles with numerous myelinated fibers, the majority of which have undergone myelin degeneration (Fig. 4, C). In the case from which the illustrations in Figure 4, C and D are taken, these bundles pass through a small ganglion which is incorporated in the internal carotid plexus. Degenerated myelinated fibers also are present in small numbers in certain other bundles in the internal carotid plexus. Marchi sections through the proximal portion of the external carotid artery exhibit a few fibers which apparently have undergone myelin degeneration. The total number of myelinated fibers in the external carotid plexus, however, is small. The evidence of fiber degeneration in Marchi preparations of this plexus, therefore, is not convincing.

Inasmuch as Marchi preparations, following section of the roots of the upper thoracic nerves, exhibit degeneration of myelinated nerve fibers in very considerable numbers in the nerves associated with the common carotid and the proximal portion of the internal carotid arteries, it seems not improbable that fibers of spinal origin extend farther cephalad, particularly along the internal carotid artery and its branches. Preganglionic spinal nerve components which extend cephalad from the upper thoracic segments, according to the best evidence available, terminate in the cervical sympathetic ganglia. The fibers which undergo degeneration distal to these ganglia, following section of thoracic spinal nerve roots, therefore, must be regarded as afferent components of the spinal nerves in question.

In order to obtain functional evidence of the existence of afferent nerve fibers along the common carotid artery, three cats were subjected to an operative procedure in which a sterile, insulated electrode was placed on this artery, with fine wires insulated by means of rubber tubes of small caliber drawn out through the skin. This device was left in position so that the nerve fibers associated with the artery could be stimulated electrically in the unanesthetized animal. In two of the cats, mild stimulation elicited reflex muscular responses, particularly in the fore limb and the upper thoracic segments, and unmistakable sensory reactions, but no evidence of intense pain. In the third cat, similar stimulation elicited marked pain reactions. When this cat was anesthetized,

slightly stronger stimulation elicited reflex muscular responses
in the fore limb and upper thoracic segments. These results clearly
indicate the existence of afferent components of the upper tho-
racic nerves along the common carotid artery. They do not indi-
cate that these components, in all cases, include fibers which nor-
mally mediate pain. Inasmuch as their stimulation elicits reflex
responses, however, they clearly constitute an afferent conduction
pathway, and their possible rôle in the production of referred pain
in the upper extremity and the upper thoracic segments is indi-
cated.

<div align="center">DISCUSSION.</div>

The data set forth in the preceding pages prove conclusively
that the nerves extending cephalad along the internal and external
carotid arteries include fibers other than those which arise in the
superior cervical sympathetic ganglion, and that these are mainly
components of the vagus and upper thoracic spinal nerves. Com-
ponents of the vagus nerve are present both in the internal and
external carotid plexuses, but are more numerous in the former
than in the latter. The spinal nerve components in question do
not traverse the upper portion of the sympathetic trunk and the
superior cervical ganglion, but ascend in the plexus on the common
carotid artery. Of those which extend into the cephalic region, the
great majority becomes incorporated in the plexus on the internal
carotid, whereas relatively few ascend in the plexus on the exter-
nal carotid artery. The anatomic relationships of the vagus and
spinal nerve components extending cephalad along the carotid
arteries are illustrated diagrammatically in Figure 5.

The vagus components in the internal and external carotid
plexuses include myelinated fibers mainly of small and interme-
diate calibers, but most of them either are unmyelinated or but
very thinly myelinated. In view of the anatomic findings of
Ranson and Michalik (1932), according to which a large per-
centage of the afferent components of the vagus nerve in the
cat, the cell bodies of which are located in the nodose ganglion,
are unmyelinated, it seems not improbable that most of the fibers
of vagus origin which extend cephalad in the internal and ex-
ternal carotid plexuses are afferent in function. The assumption
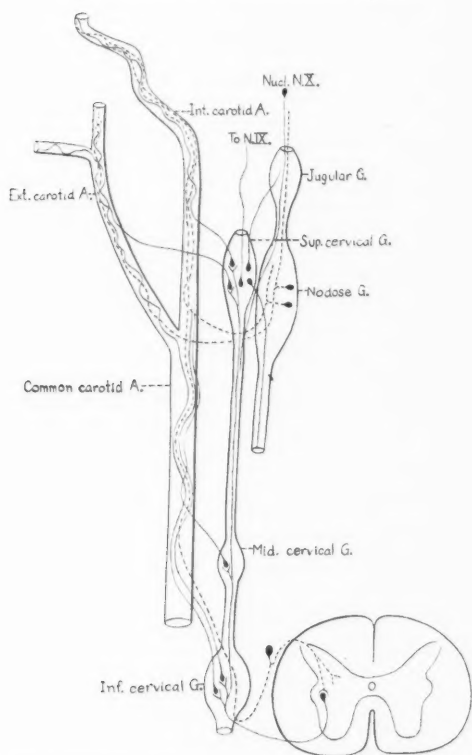
Fig. 5. Diagrammatic representation of the anatomical relationships of the vagus and spinal nerve components which extend cephalad along the common, internal and external carotid arteries.

that afferent fibers of vagus origin are present in these plexuses also is supported by clinical data (Fay, 1932).

Some of the fibers which remain intact in the plexuses on the common, internal and external carotid arteries following extirpation of the superior cervical sympathetic and nodose ganglia doubtlessly arise in the middle and inferior cervical sympathetic ganglia. Others, as is demonstrated by the presence in these plexuses of degenerated fibers in Marchi preparations following section of the roots of the upper thoracic nerves just distal to the spinal ganglia, are components of the thoracic spinal nerves.

Inasmuch as preganglionic components of these nerves can effect functional connections only in the sympathetic ganglia, the fibers in question must be regarded as afferent spinal nerve components which traverse the sympathetic trunk in the upper thoracic and lower cervical segments and join the plexus in the common carotid artery.

The existence of a small number of fibers in the internal and external carotid plexuses which apparently failed to undergo degeneration following ligation and section of the common carotid artery and division of the entire carotid sheath, in addition to extirpation of the superior cervical and nodose ganglia, seems to indicate that a few fibers join these plexuses from sources other than the sympathetic trunk and the vagus and thoracic spinal nerves. In some instances a small aggregate of ganglion cells has been observed in the internal carotid plexus near the origin of the internal carotid artery. No ganglion cells have been observed in the external carotid plexus. The existence of ganglion cells along the external carotid artery, however, is not precluded. The existence of some intact fibers in the plexuses on the internal and external carotid arteries, following the operative procedure indicated above, probably can be explained most satisfactorily on the assumption that they arise from sympathetic ganglion cells which are not incorporated in the superior cervical sympathetic ganglion. On the other hand, the existence in these plexuses of components of the upper cervical spinal nerves and fibers arising in the jugular ganglion is not precluded.

The carotid sinus nerve is present in many of our sections through the proximal portions of the internal carotid arteries. This nerve, as previously stated, is composed mainly of myelinated fibers. Its branches terminate in the wall of the carotid sinus and do not mingle freely with the rami which constitute the internal carotid nerve. The carotid sinus nerve has been recognized in the sections but has played no part in the data set forth in this paper.

Demonstration of the existence of afferent vagus components in the internal and external carotid plexuses affords an anatomic basis for the assumption, based on clinical observations, that sensory impulses arising in the area of distribution of the trigeminal

and facial nerves may reach the brain stem through fibers which are not incorporated in the trigeminus and the nervus intermedius. Demonstration of the existence in the plexuses on the common and internal carotid arteries of afferent components of the thoracic spinal nerves and the probable existence of such fibers in the external carotid plexus affords an anatomic explanation of the theory advanced by Fay (1932), on the basis of clinical observations, that there exists along the carotid arteries a descending conduction pathway from the face to the upper thoracic segments of the spinal cord.

The fact that direct stimulation of the descending fibers along the common carotid artery elicits reflex responses in the fore limb and the upper thoracic segments suggests that these fibers play an essential rôle in the production of referred pain in these parts of the body, due to lesions in the orbit, the mastoid area and the mucous membranes of the nose and paranasal sinuses. According to the current teaching, the somatic areas in which referred pains are localized always fall within the segments which are supplied by those spinal nerves which also supply afferent fibers to the area in which the lesion responsible for the pain is located. According to the older theories of referred pain, exaggerated stimulation of the afferent fibers which conduct impulses from the site of a visceral lesion into the spinal cord results in hyperirritability of the afferent neurons in the segments of the cord in question. The neurons which normally conduct cutaneous impulses to the brain, consequently, become activated. The resulting sensations are referred by the brain, not to the site of the lesion, but to the cutaneous area connected, through its afferent innervation, with the neurons in question in the spinal cord.

Although these theories afford a plausible explanation of the mechanism of referred pain, they do not adequately take into consideration certain reflex phenomena which commonly are associated with referred pain, viz., vasoconstriction, activation of sweat glands, cutis anserina and muscular hypertonus, in the painful area. These phenomena represent reflex responses elicited by afferent impulses arising at the site of the lesion. Hypertension or spastic contraction of the smooth muscle in vessel walls is known to be a major factor in the production of pain referable

to blood vessels either by reason of its direct effect on sensory
receptors or the consequent ischemia of the adjacent tissues.
Hypertension of skeletal muscles, likewise, may give rise to pain
by reason of its stimulating effect on sensory receptors in the
muscle tissue. Since painful stimuli are the most provocative
causes of reflexes, the vessels in a peripheral area of vasoconstric-
tion tend to remain constricted and acutely tender hypertonic
muscles tend to remain hypertonic for some time after the
exaggerated stimulation in the area of the primary lesion has sub-
sided. The actual stimulation of pain receptors in the area in
which the referred pain is localized, therefore, must be regarded
as an important factor in the production of the pain. This conclu-
sion also is supported by the results of clinical and experimental
studies (Weiss and Davis, 1929; Morley, 1929; Rudolf and Smith,
1930), which have demonstrated that referred pains associated
with various visceral diseases may be alleviated or completely
abolished by local anesthesia of the painful area.

In view of the anatomic and physiologic findings set forth in
the present paper and the above consideration of the mechanism
of referred pain, it is apparent that pains localized in the upper
extremity and the upper thoracic segments, which are associated
with lesions in the orbit, mastoid area and the mucous membranes
of the nose and paranasal sinuses, conform to the generally recog-
nized principles of the localization of referred pains. The data
presented support the assumptions that such referred pains are
mediated through the upper thoracic nerves and that afferent
impulses arising in the primary lesions are conducted from the
sites of these lesions into the spinal cord through afferent compo-
nents of these nerves which traverse the nerves associated with
the common, internal and external carotid arteries.

SUMMARY.

1. Extirpation of the superior cervical sympathetic ganglion
results in degeneration of many nerve fibers in the internal and
external carotid plexuses, but leaves a goodly number intact.

2. Extirpation of the entire cervical sympathetic trunk results
in degeneration of a larger proportion of the fibers in the internal
and external carotid plexuses, but many still remain intact.

3. Section of the upper four or five cervical spinal nerves, in addition to extirpation of the cervical sympathetic trunk, exerts no appreciable influence on the resulting nerve fiber degeneration in the internal and external carotid plexuses.

4. Extirpation of the superior cervical sympathetic ganglion, ligation and section of the common carotid artery and division of the carotid sheath results in nerve fiber degeneration in the internal and external carotid plexuses approximately equal to that resulting from extirpation of the entire cervical sympathetic trunk.

5. Extirpation of the superior cervical sympathetic and nodose ganglia results in more extensive nerve fiber degeneration in the internal and external carotid plexuses than extirpation of only the superior cervical sympathetic ganglion.

6. Ligation and section of the common carotid artery and division of the carotid sheath, in addition to extirpation of the superior cervical sympathetic and nodose ganglia, results in almost complete nerve fiber degeneration in the internal and external carotid plexuses.

7. Section of the roots of the upper four or five thoracic spinal nerves just distal to the spinal ganglia results in degeneration of a goodly number of nerve fibers along the common carotid artery and in the internal carotid plexus and probably some fibers in the external carotid plexus.

8. The above results support the conclusion that components both of the vagus and upper thoracic spinal nerves extend cephalad in the internal and external carotid plexuses. These components are mainly, perhaps exclusively, afferent in function. The vagus components afford a possible pathway for afferent impulses from the area of distribution of the fifth and seventh cranial nerves into the brain stem. The spinal nerve components afford a conduction pathway for afferent impulses from the same area into the upper thoracic segments of the spinal cord. Both these pathways may play a rôle in certain cases of atypical facial neuralgia.

9. Demonstration of the existence of afferent components of the upper thoracic spinal nerves in the carotid plexuses affords the anatomic basis for a rational explanation of the localization

in the upper extremity and the upper thoracic segments of referred pains resulting from lesions in the orbit, the mastoid area and the mucous membranes of the nose and paranasal sinuses.

REFERENCES.

Cannon, B. (1933) : A Method of Stimulating Autonomic Nerves in the Unanesthetized Cat, With Observations on the Motor and Sensory Effects. Am. Jour. Physiol., 105, 366-372.

Carmichael, E. A., and Wollard, H. H. (1933) : Some Observations on the Fifth and Seventh Cranial Nerves. Brain, 56, 109-125.

Davis, L., and Pollock, L. J. (1932) : The Rôle of the Sympathetic Nervous System in the Production of Pain in the Head. Arch. Neur. and Psychiat., 27, 282.

Fay, T. (1932) : Atypical Facial Neuralgia, a Syndrome of Vascular Pain. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 41, 1030-1062.

Foerster, O., Altenburger, H., and Kroll, F. W. (1929) : Ueber die Beziehung des Vegetativen Nervensystems zur Sensibilitat. Ztschr. f. d. ges. Neur. u. Psych., 121, 139-185.

Helson, H. (1932) : The Part Played by the Sympathetic System as an Afferent Mechanism in the Region of the Trigeminus. Brain, 55, 114-121.

Morley, J. (1929) : Afferent Impulses From the Skin in the Mechanism of Abdominal Pain. Lancet, 217, 1240-1244.

Ranson, S. W., and Mihalik, P. (1932) : The structure of the Vagus Nerve. Anat. Rec., 54, 355-360.

Rudolf, R. D., and Smith, A. G. (1930) : Visceral Pain. Am. Jour. Med. Sci., 180, 558-568.

Sluder, G. (1920) : Headaches and Eye Disorders of Nasal Origin. C. V. Mosby Co., St. Louis.

Terracol, J. (1932) : Diagnostic Interest of Referred Pains in the Territory of the Cranial Nerves. Rev. d'oto-neuro-opht., 10, 246.

Weiss, S., and Davis, D., 1928. The Significance of the Afferent Impulses From the Skin in the Mechanism of Visceral Pain. Skin infiltration as a useful therapeutic measure. Am. Jour. Med. Sci., 176, 517-536.

VI.

HYPERTONIC MUSCLES OF THE NECK AS A CAUSE OF HEADACHE.*

WILLIAM MITHOEFER, M. D.,

CINCINNATI.

Headache, as we all know, is one of the most frequent symptoms complained of by patients visiting the otolaryngologist. A patient with an acute disease of the upper respiratory tract, who complains of headache, usually shows upon examination a manifest reason for this complaint. It is otherwise, in many instances, however, in patients who are chronic sufferers. It is in this class of patients especially, where a definite schedule should be followed in making an examination, and the muscles of the neck should be included in the routine.

There are many otolaryngologists who, in the course of their examination, do not give the muscles of the neck the attention they deserve, so that in consequence many patients have been subjected to repeated operations without obtaining relief of the headache, the sole cause of which was to be found in the structural or functional changes in the neck muscles.

There is plenty of evidence to be found in the records of our office to prove that such is the case. Certain it is, in many instances, that the changes in the muscles are caused by a focus of infection in the tonsils, the teeth or the nasal accessory sinuses, and that after the removal of the infection there is, in many cases, a disappearance of the occipital headache. There is, unfortunately, a large number of patients who do not find relief after removal of a focus of infection of the ear, nose and throat, and in whom the changes in the muscles are caused by some constitutional derangement in the form of a dysfunction of the endocrine system, gouty arthritic diathesis, chronic intestinal toxemia, gall bladder infection, etc. Many patients have hypertonic muscles of the neck

and never suffer with headaches. Others with a mild hypertonus acquire a severe headache upon the slightest exertion. Physical exercise does not bring on an attack as quickly as does the bending forward of the head during work. Bookkeepers, typists, proofreaders and dressmakers are very susceptible. A long motor trip, nervous excitement, and mental strain are also predisposing factors. A large number of patients are women in whom the headaches begin at the time of the menopause. It must not be forgotten that an occipital headache may very often be present in patients suffering with a muscular imbalance of the eyes.

Various authors use different names in describing the changes that have taken place in the muscles of the neck. Myalgia, indurated headache, nodular headache, muscular headache and muscular rheumatism of the neck are terms which have been used. Several investigators have made a pathologic examination of the excised parts of the muscle. They were unable, however, to explain the true nature of the affection.

The pain usually begins in the occipital region and may radiate towards the back, the shoulders and over the head to the frontal region. It is present very often upon awakening, in this way simulating headache as the result of nasal accessory sinus disease. Very often the headache is more pronounced when the patient has slept longer than usual. During the day the muscular exercise diminishes a sufficient amount of tension so that towards evening the headache has a tendency to disappear, again simulating nasal accessory sinus disease. This is true, however, only in mild cases. When the patient, as so often happens, remains bedfast, the headache continues unabated and may persist for weeks or even months.

In severe cases vertigo, vomiting and earache may be prominent symptoms. The vertigo and vomiting may be accounted for by the irritation of the nerve endings of the auricular branch of the vagus as it passes through the hypertonic sterno-cleido-mastoid muscle; the earache from irritation of the temporal branches of the auriculotemporal nerve which sends branches to the auricle and external auditory canal. It is difficult to say whether tinnitus is the result of an irritation of the auriculo-

temporal nerve or is produced by the constant pull on the auricle by the small muscles of the ear as the result of their intimate connection with the occipitofrontalis aponeurosis and the sterno-cleido-mastoid muscle. A constant pull on the auricle and skin lining the external auditory canal may exert some influence on the tympanic membrane, and in consequence of this, be the cause of a tinnitus. It can readily be understood that structural changes in the muscles, in the form of nodules and induration, are very apt to cause an irritation of nerves in the same way that sciatica may be the result of an extension of a myositis to the sciatic nerve. A muscle which has become involved in such a manner becomes hypertonic, and in this state produces severe pain in the head. It is impossible to state whether the presence of the nodules in the muscles or the hypertonicity itself is the cause of the headache. Certain it is that many patients in whom, upon palpation, no nodules can be felt, and whose muscles are hard and hypertonic, suffer as intensely as those in whom definite areas of induration are felt at the time of the examination and treatment. We may readily deduct, therefore, that functional changes alone may account for the hypertonic state of the muscles.

Another cause of hypertonic muscles of the neck is the presence of a latent arthritis. The joints most frequently involved are the sternoclavicular, the acromioclavicular and the cervical vertebræ. The patient may or may not have an arthritis of other joints of the body. In some of the severe cases of muscular headache, a skiagram has shown marked changes in the joints, and it is worthy of mention that patients in whom the joints present pathologic changes had little or no headache after the removal of the foci of infection. When a joint becomes arthritic, the muscles attached to that joint may become hypertonic. Muscles most frequently involved are the sterno-cleido-mastoid, the trapezius and the splenius capitus. Physiology teaches us that a muscle is always in a state of slight tension. It is obvious that if a muscle were lax, considerable energy would be wasted to overcome this laxity at the moment of contraction. Under normal conditions the tendinous attachment of a muscle offers a certain amount of resistance during contraction. If this resistance be increased as a result of a latent arthritis of the joint to which the tendon

is attached, there will occur an increased tension of the entire muscle—in other words, a hypertonic state of the muscle will ensue. As the circulation of the blood through a muscle depends on the contraction and relaxation of that muscle, it may readily be seen that if a muscle becomes hypertonic, relaxation disappears, the circulation is disturbed, and hyperemia with consequent thickening of the muscle fibers and occasionally the formation of nodules results.

It is important to remember that the occipitofrontalis muscle blends posteriorly at the superior curved line of the occipital bone with the trapezius, sternomastoid and splenius capitus, and anteriorly with the orbicularis palpebrarum and the corrugator supercilii. There is also an intimate connection between the fibers of the temporal muscle and those of the occipitofrontalis. It is difficult to say whether the headache that results is a true muscular pain or whether it is directly due to the involvement of the nerves, such as the occipital and supra-orbital, in the thickened fibers of the muscles which they pierce. In fact, Peritz claims that a nerve may be painful on pressure, not as the result of a primary disease of the nerve itself, but secondary to increased muscular tension. The question arises, why does not the pain assert itself in the region of the joint affected with a latent arthritis, instead of expressing itself in the form of a headache? This can probably be explained by the fact that in a latent disease of a joint the muscle attached to the joint is more painful than the joint itself. Furthermore, it must not be forgotten that there is an intimate connection between the fibers of the cervical nerves and the fifth nerve, both in their deep origin and in their superficial branches. It is therefore possible that an irritation of the great occipital nerve, as it pierces a hypertonic sternomastoid muscle may produce a pain radiating along the course of the trifacial nerve.

The painful areas which may be present are the following: Region of sternoclavicular joint, sterno-cleido-mastoid muscle, acromioclavicular joint, triangular area between the tip of the mastoid process and the angle of the jaw, region of the occipital nerve, cervical vertebræ, pulley of superior oblique muscle, trapezius muscle, temporal muscle.

One of the most important and constant symptoms is the pain on pressure over the sternoclavicular joint on its posterior aspect. In order to determine satisfactorily the presence of a painful clavicular joint, the index finger is placed as deeply as possible in the jugulum and pressure made from within outward. A distinct infiltration may occasionally be felt in the region of the joint. In some patients pressure is so painful that manipulation becomes almost impossible. The tendinous attachment of the sterno-cleido-mastoid may also be very painful on pressure, and it has been noticed, during the course of treatment, that with the subsidence of pain in this region, there was also a disappearance of the headache. It is rather disconcerting when a patient with a chronic discharging ear suffers with hypertonic neck muscles, and when there is present a painful area at the tip of the mastoid. We must not allow this fact to influence us in making the indication for a radical mastoid operation, inasmuch as the pain may still be present after the operative procedure. This likelihood must be explained to the patient before operation. If this is done, the operator may be saved considerable embarrassment. The following case will illustrate this point.

Female, age 24, has had a discharging ear since childhood. For two months she has had a constant pain over the tip of the mastoid process on the affected side, which is increased on pressure. There is present a perforation of Shrapnell's membrane, with a small granulation springing from the upper posterior edge. The patient was told that the pain in the mastoid was caused by the chronic ear disease, and, in consequence, consent for operation was readily given. Immediately following the radical mastoid operation the pain disappeared and healing was complete in nine weeks. Three weeks later, or in other words, three months after operation, the patient again presented herself, complaining of the same pain in the region of the mastoid for which she consulted us at her first visit. It was then that careful palpation of the muscles of the neck demonstrated an extreme hypertonic condition of the sterno-cleido-mastoid, with a latent arthritis of the sternoclavicular joint. The only explanation that could be given was that at the time of the operation the division of the sterno-cleido-mastoid fibers at the tip of the mastoid was sufficient to

relax the hypertonic muscle, and that after a certain length of time had elapsed the fibers again became firmly adherent, thereby causing the muscle to become hypertonic once more. Halle reports a similar case.

Another patient with severe pain in the region of the mastoid, and in whom there was a discharging ear with a large central perforation, was referred to us for a radical mastoid operation. In this case, however, we did not operate, but instead instituted a course of massage which relieved the patient in three days. It is worthy of mention that this patient had been bedfast for one week, was not relieved by the administration of morphia and was brought to the hospital in an ambulance. The relief of this patient was one of the most dramatic results I have ever seen following massage of hypertonic neck muscles, inasmuch as there was almost complete relief after the first massage.

In order to make a satisfactory examination of a patient suffering with hypertonic neck muscles, it is necessary to have the muscles relaxed as much as possible. The head is therefore bent backwards in examining the posterior group, and forward, with the chin turned toward the side being examined, when the sterno-cleido-mastoid muscle is being palpated. It is very necessary that all painful areas be found at the time of examination, so that massage may be instituted in these regions. The region of the superior oblique and temporal muscles must not be overlooked. The same is true of the occipital nerve at a spot half way between the mastoid tip and the occipital protuberance. The tactile sense becomes very much exaggerated if the resistance of the skin is removed by the application of a lubricating jelly. If the patient is examined when the headache is not present, the muscle feels thicker than normal, in other words, one is apt to term it a well developed muscle. The opposite is true during an attack of headache. The muscle becomes very tense and light palpation is painful.

In the treatment of hypertonic muscles of the neck, the best means at our command is massage. Mild cases are usually relieved after a few massages, whereas chronic cases may require from twenty to thirty before any apparent beneficial results are ob-

tained. A routine method of massage should be used in every case. With the patient in a sitting position, palpation of the head and neck is first made in order to determine the presence of painful areas. A lubricating jelly may be used, if desired. To massage the neck in an intelligent manner, we must take into consideration the direction of the muscle fibers. Two movements of massage are employed, the longitudinal along the course of the muscle fibers, and the transverse massage, across the fibers. Longitudinal massage relaxes the muscle fibers and often gives immediate relief, but it is not as beneficial as the transverse massage which usually causes a momentary increase of tension. The reaction which occurs after each transverse massage of the muscle is followed by a relaxation, which, after a sufficient number of massages, reduces enough of the hypertonus to relieve the patient of headache. The best method of using transverse massage is to grasp the muscle repeatedly with the thumb and index finger. This transverse massage should be applied chiefly in the region of the painful areas, for as these areas become less painful, the headache usually disappears.

The regions of the occipital nerve, superior oblique muscles, temporal muscles and various joints require a different form of massage. In these areas we use a treatment which, for want of a better name, we have termed pressure massage. The thumb is pressed firmly against the painful area and pressure is continued for about one minute. This is repeated at least three times, and is supplemented by a gentle longitudinal massage of the region being pressed upon. There is always a certain amount of pain with every massage, but occasionally a case is seen where the mildest massage produces such excruciating pain that the patient refuses further treatment. If the massage is too painful, little good can be accomplished. It is therefore advisable to instruct the masseur to keep the pain during massage within certain bounds, and if this is done it will be found, within a few days, that the muscles will not be sensitive to touch, and a more energetic massage may be used. The rule is, therefore, the more painful the muscle is to the touch, the milder the massage. Massage given more than three times a week is not necessary. When relief has taken place, an occasional massage every two weeks

over a period of a year or more is advisable in patients who are chronic sufferers. The patient must be instructed not to take a continued rest after each massage. It is far better, after a rest of several hours, to take a long walk. In fact, all patients with muscular headaches should be advised to walk from three to five miles daily. Slow walking is not as beneficial as fast walking, for in the latter the movements of the muscles have a tendency to reduce a certain amount of the hypertonus. It is scarcely necessary to remark that all foci of infection that may be the cause of the latent arthritis must be eliminated before instituting a course of massage.

In addition to the massage, the application of prolonged infrared heat has been found of great benefit. The time of exposure should be at least forty-five minutes to an hour. As a matter of fact, mild cases may be relieved by radiant heat alone.

When there is present a gouty arthritic diathesis, it becomes necessary to have a general massage twice a week. The masseur will tell you that these patients have many painful spots in the muscles in various parts of the body. This is not always the result of a focus of infection, but may be caused solely by a faulty metabolism from improper diet and lack of exercise. It becomes necessary, therefore, in most cases to insist on free elimination in the form of catharsis, sweat baths, meat-free diet, the drinking of at least eight or ten glasses of water a day, and exercise in the open air, in the form of walking.

Atophan is a valuable drug in some cases and should be given three times a day, after meals, for three successive days each week. Another valuable adjunct in the treatment is the administration of thyroid and ovarian extract. The latter is given intramuscularly, three times a week, and the former in increasing doses until the tolerant dose is reached.

In conclusion, may I again impress upon you the importance of the symptom-complex arising from hypertonic neck muscles, and venture the remark that no examination of a patient suffering with headache, especially one of the chronic type, is complete without giving these muscles the attention they deserve. Our

experience has taught us that the results obtained from proper treatment of hypertonic neck muscles are most satisfactory.

19 GARFIELD PLACE.

BIBLIOGRAPHY.

Halle: Monatschrift f. Ohrenheilkunde, 1911.
Auerbach: "Headache," 1912.
Mueller, A.: "Muscular Headache," 1911.
Peritz: Berlin Klin. Wochenschr., 1907, No. 30.

VII.

ADVANCED METHODS IN THE SURGICAL TREAT-
MENT OF FACIAL PARALYSIS.*

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NEW YORK.

I had expected, when I accepted your invitation, to present a paper on a limited aspect of facial paralysis, dealing with the pathology of the lesion. Unfortunately the histologic and pathologic studies of some twenty-odd primates, which clinically had inspired me to this effort, have not sufficiently advanced to permit me to make the demonstration I had anticipated. I have, therefore, decided to offer a short resumé of the changes in the surgical technic of repair of the facial nerve which increasing experience has evolved, and, at the end, to offer some clinical reasons for a proposed surgical treatment of certain cases of Bell's palsy. I hope in the near future to present some histologic support for this proposition.

It is now well over 100 years since Sir Charles Bell so accurately described the anatomy and physiology of facial expression and explained the cause of facial palsy.[1]

Although a paralysis of the facial nerve, from any cause, has, since that time been called Bell's palsy, there is now a tendency to limit this nomenclature to the cases which are not due to suppuration in the temporal bone or to accidental injury of the nerve in the surgical treatment of mastoiditis. In other words, we now think of "Bell's palsy" as either a refrigeration or a toxic infection of the nerve; and of "facial paralysis" as a palsy resulting from some other cause, such as necrosis, or suppuration in the temporal bone or operative trauma, etc.

Although the nature of the lesion was well recognized, no effort to surgically relieve the condition was made until 1879. There is then only a meager account of an effort of Drobnick to anastomose the spinal accessory to the facial in the neck. The report

was made by one of his confreres and the final results are not recorded. We must, then, attribute to Sir Charles Ballance of London the first anastomosis of the spinal accessory to the facial in which the details were published in 1895. Following this work, the operation of anastomosis of the facial with the neighboring nerves in the neck (spinal, accessory, hypoglossal, descendens noni, glossopharyngeal)—was employed by many eminent surgeons and the method perfected to a point which may well be called brilliant. Nevertheless, the best successes were always marred by the fact that there was no emotional response in the facial expression; and voluntary changes were usually accompanied by associated movements.

Looking back on the progress of surgical intervention with the lesion, it seems quite remarkable that, although direct repair of the lesion in the injured nerve was attempted by Ballance in 1894, by Stacke in 1903, by Alt in 1908, by Marsh in 1900, and again in 1908, by Sydenham in 1909, by Ney in 1922, by Bunnell in 1925-30, by Martin in 1931,[2] the repair by anastomosis with another nerve in the neck remained the operation of choice during all these years.

Two years ago I had the pleasure of appearing before the otolaryngologic section of this college, presenting the outcome of a year of experimentation on animals by Sir Charles Ballance and myself, in which we had demonstrated that autoplastic grafts of any length, whether reversed or unreversed, would restore the function of the injured facial nerve. Moreover, this method of repair eliminated the associated movements which resulted from anastomosis with other nerves, and, at the same time, brought about emotional facial expression.

Since that time I have operated upon fifty cases of facial paralysis resulting from various lesions. I have learned many lessons from this experience, and should like to present moving pictures of selected cases from this group which will accentuate some important points in the method.

Preconceived theories regarding the nature and extent of the injuries which probably would be encountered in a large series of cases were rapidly modified by clinical experience. For example, Sir Charles and I had thought that the extent of the injury

Case 1. Baby 9—Facial graft, 27 mm. From anterior respiratory nerve of Bell. Before operation.

Case 1. Baby 9—14 months after operation.
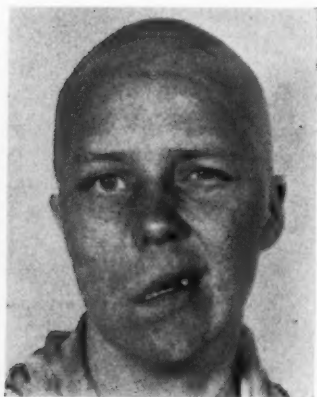
Case 2. Mrs. Agnes B.—Facial graft, 30 mm. From anterior respiratory nerve of Bell. Before operation.

Case 2. Mrs. Agnes B.—26 months after operation.

to the nerve would hardly exceed 5 mm. in the majority of cases. This conjecture was due to the fact that in all of the large number of surgical repairs by anastomosis, the site of the injury to the facial nerve had never been investigated. The few cases reported by Ney and others had required only a decompression. Martin's case suggested a shortening of the route to gain a few mm. In my series of fifty cases there have been thirty-five in which the injury required a graft. Of these there was one of 7 mm., two of 9 mm., and one in which removal of the parotid gland had caused a gap, the repair of which required four measured grafts aggregating 150 mm. Of the remaining thirty-two none was under 15 mm., and the average length of all was 20 mm.

This rather astounding experience, at the very outset confronted us with a serious question as to the nerve which should be employed as a graft. We started out by using Bell's anterior respiratory nerve. In our animals we never had used more than 15 mm. We had succeeded in every instance in uniting the severed ends of the respiratory nerve, with final restoration of function of the serratus magnus muscle. It was obviously quite impossible to do this when such great lengths were excised. We sought a motor nerve the loss of the muscle which it controlled would be of less consequence. The intercostal nerves were used with success in many cases. The danger of the injury to the pleura in the removal of these nerves, however, made us look elsewhere. We had demonstrated that nerve trunks of sensory nerves were just as efficient as motor nerves, and, after trying several we fixed on the anterior femoral cutaneous. Any length required can be secured from this nerve, and the destruction of it results only in a small area of anesthesia in the thigh, which I have found is subsequently largely restored by collateral branches.

We had also thought that suppurating fields were a contraindication to the employment of grafts. In the first case which I show you we employed 26 mm. of graft carried through a field bathed in pus. In my subsequent experience grafts have been successfully placed in purulent fields in more than half of all the grafted cases. It can be confidently stated that an infected field is not a contraindication of the employment of nerve grafts.

ARTHUR B. DUEL.

Case 3. Miss C. C.—Facial graft, 20 mm. From intercostal nerve. Before operation.

Case 3. Miss C. C.—17 months after operation.

Case 4. Miss W. S.—Facial graft, 16 mm. Double strand. From Intercostal nerve. Before operation.

Case 4. Miss W. S.—16 months after operation.

We had also thought that the region surrounding the ends of the graft must be especially dry so far as bleeding is concerned, and hours have been spent in some of my cases in accomplishing this before the graft was inserted. We now have learned that, if the field can be completely dried temporarily, and the ends of the graft closely approximated to the distal and proximal segments, the clot (forming the ooze around the nerve) makes the best fixation of the graft and probably assures success. It is as if the field of operation were embedded in gelatin. The blood clot should not come between the ends of the implant, but, as soon as the ends are closely approximated to the distal and proximal segments, may well surround the whole replacement.

While we obtained encouraging successes (in these early cases repaired by fresh grafts) the long period which elapsed between the successful implantation and the beginning responses in the muscles led me to make some further animal experimentation. The history is now comparatively old, of successful shortening of the time and improvement in the quality of the repair by the employment of grafts which were taken from nerves in which Wallerian degeneration had been allowed to take place in situ for from two to three weeks. I shall show you some pictures of cases in which the first response came through (where grafts degenerated in situ had been employed) in one-third of the time required in the earlier cases where fresh grafts had been used.

Another feature of this experience may interest you. Owing to the sudden wave of propaganda resulting from the reports of some of these cases, a large influx of patients was thrust upon me. At one time there were twelve cases at the Manhattan Eye, Ear and Throat Hospital simultaneously. The preparation (by degeneration of the nerve in situ) before the material could be used as a graft, required three weeks. It occurred to me that inasmuch as the femoral cutaneous nerve which we were employing was such a long one, it might be possible to employ heteroplastic grafts. Discussing the matter with Dr. Eggston, the pathologist, he suggested that if I were to attempt this it might be well to employ such grafts only from cases in which the blood type of the donor and recipient was the same; on the same ground that blood transfusion and skin grafts (in such cases) had been found to

Case 5. Miss M. Y.—Facial graft, 7 mm., double strand. From intercostal nerve. Before operation.

Case 5. Miss M. Y.—10 months after operation.

Case 6. Mr. A. J.—Facial graft, 12 mm. From anterior femoral cutaneous nerve (prepared by degeneration in situ). 10 months after operation. (In repose.)

Case 6. Mr. A. J.—10 months after operation. (In action.)

be much more successful. Accordingly, we ordered that all pa-
tients with facial palsy should be blood typed on their entrance.

On one occasion I found that a patient whose nerve had been
prepared and who was all ready for operation was the same type
as two others who had just been admitted. I operated upon the
three cases, using the prepared nerve from one. The grafts in all
were successful. One of the heteroplastic recipients is nearly com-
pletely recovered. The other two are beginning to use some of
their facial muscles and promise eventually to make a fairly good
recovery. I have since found two other patients in whom I have
used heteroplastic grafts successfully. The significance of this
employment of heteroplastic grafts in individuals of the same
blood type may be far-reaching in neurosurgery if we are ever
unfortunate to have another world war.

I should like to speak, from a clinical standpoint, on the sub-
ject to which I originally intended to confine myself in this dis-
course. As you all know, the nontraumatic refrigeration or toxic
types of neuritis which are designated by the term Bell's palsy
furnish by a large majority the total number of cases of facial
paralysis. The percentage of recovery in these cases is large. I can-
not give exact statistical percentages, and I doubt if anyone could
be exact. Suppose we say, roughly, that 90 per cent of the cases
in from six to eight weeks make a recovery which is perfect. This
leaves a small percentage (which, nevertheless, in the aggregate,
furnishes a very large number of cases) in which recovery is so
incomplete as to leave the victims with a very disfiguring defor-
mity, often accompanied by a facial spasm which is very distress-
ing. The lesion which is back of all this is similar to the lesion
which produces a neuritis in any nerve. The difficulty in
neuritis of the facial nerve is that the swelling incident to the
infection, occurring in a nerve which is surrounded by a tube of
ivory-like bone, produces a compression of the neurons for a
sufficient period to stop the function of the nerve. Now in the
severe types this pressure amounts to a trauma quite as extensive
as that in accidental operative injuries. In such cases Wallerian
degeneration of all nerve cells distal to the point of this squeeze
takes place. The analogy is quite apparent. Where a slight acci-
dental trauma during operation takes place, the recovery of the

Case 7. Miss F. A.—Facial graft, 10 mm. From anterior femoral cutaneous nerve (prepared by degeneration in situ). Before operation.

Case 7. Miss F. A.—9 months after operation.
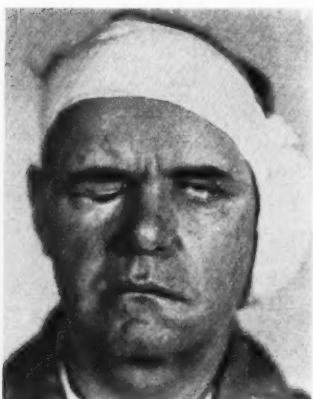
Case 8. Mr. M. F.—Facial graft, 7 mm. (double strand heteroplastic). From anterior femoral cutaneous nerve (prepared by degeneration in situ). Before operation.
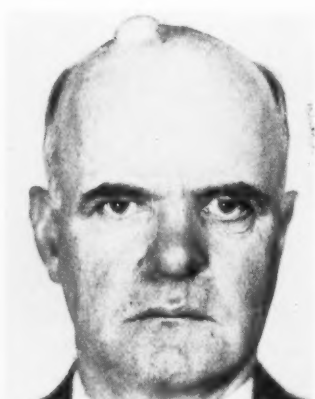
Case 8. Mr. M. F.—8 months after operation.

nerve may be perfect or almost perfect. When this happens we feel that we might have been quite justified in leaving the case under observation. However, when such an accident happens the uncertainty of how extensive the inflammatory squeeze may be, and the uncertainty, therefore, of what the extent of the recovery may be, has convinced me that any facial paralysis following accidental operative trauma should be uncovered at once, and the case treated according to the lesion that is found, varying from simple drainage to slitting of the nerve sheath, or immediate repair of neurons by autoplastic grafts.

In every instance immediate investigation followed by appropriate treatment insures a much nearer perfect result.

Discussing this problem with Sir Charles during the two years in which we were intimately connected in the animal experimentation, he often said, "These cases of Bell's palsy which make a partial recovery and then go on without further improvement for weeks or months, ought to be uncovered early and decompressed to relieve the squeeze at that stage. Such cases (unless they are appropriately treated) in all probability will go through life with a grotesque deformity." In principle I thoroughly agree with this opinion. I realize, however, there is great difficulty in convincing those who come in contact with these cases that this is sound judgment. The trouble is that those who see and treat these cases are very loath to subject them to such a radical procedure as uncovering the nerve and slitting the sheath in the hope that the recovery will be sufficiently good to warrant it. The thought of subjecting a nerve to a trauma which is actually the cause of so many palsies of the face, when recovery has already partially taken place, is too appalling. Realizing this, and hoping that I might correct an erroneous belief, I did a series of experiments on rhesus monkeys in which I uncovered the nerve and split the sheath. Ten monkeys on which I split the sheath of the nerve over an area of 10 to 15 mm. were examined daily afterward over a long period. Nine of the ten suffered no facial paralysis, no Wallerian degeneration, no loss of faradic response. One of them gradually lost faradic response so that there was evidence of complete Wallerian degeneration at the end of nine days. Seventeen days later, however, faradic response began

Case 9. Mary A.—Decompression. Before operation. (In action.)

Case 9. Mary A.—3 months after operation. (In action.)

Case 10. Miss E. S.—Decompression 6 months after operation. (In repose.)

Case 10. Miss E. S.—6 months after operation. (In action.)

again, and in six weeks the face had apparently recovered. This case, undoubtedly, had suffered an injury to the neurons by the process of splitting the nerve sheath, or had undergone a slight infection. In either case, however, he had recovered quite as rapidly as any complete case of Bell's palsy. The other nine seem to prove that the actual trauma involved in splitting the sheath, when carefully done, does not cause facial palsy.

I then induced facial palsy in a series of monkeys by exposing and freezing the nerve with ethyl chloride. One case was left without splitting the sheath; all the other cases had the nerve sheath split open over the frozen area and a few mms. distal and proximal to that area. The cases in which the nerve sheath was split recovered facial movements in one-half the time that it required for recovery in the other cases.

The same experiment was made on a series of monkeys in which facial palsy had been induced by the injection of 90 per cent alcohol into the nerve. In a similar way the cases in which the nerve sheath was split over the area of toxic involvement recovered twice as rapidly as those in which the sheath was not split.

Fortified by this experience, I summoned up sufficient courage to split the sheath of the nerves of several cases of Bell's palsy in which the recovery was very incomplete and had remained unchanged for many years. In every instance, despite this long period of inactivity, the relief of the pressure by splitting the nerve has resulted in a very marked improvement.

Accompanying is the picture of one case in which a double Bell's palsy, rendering the patient very grotesque, has been relieved to the point of making equal and synchronous movements of both sides of the face possible.

Now, arguing from this, we might theoretically say that cases of Bell's palsy which have failed to improve over a long period (let us say, several months) might take the chance of a very much greater improvement by being subjected to surgical intervention (decompression of the nerve by splitting the sheath) rather than to go on with almost certain grotesque deformity throughout their lives. I realize that this is almost heretical, certainly very audacious, but I am convinced that eventually it will be the accepted procedure. It will necessitate a careful observation of a large

Case 11. Miss N. S.—Bell's palsy.
Right side. 6 months. Left side.
10 years. Double decompression.
Before operation.

Case 11. Miss N. S.—17 months
after operation.

number of cases so that one will be able to say "such and such a
case which has now failed over such and such a length of time
to make any definite improvement will probably go on for the
rest of his life without recovery if left unoperated upon; that
such a case will probably make a very definite—possibly almost
complete—recovery, if operated upon early."

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REFERENCES.

Bell, Sir Charles: "The Anatomy and Physiology of Facial Expression."
6th edition. 1872. Henry G. Bohn, publishers.
Ballance, C.: "Surgery of the Temporal Bone." New York: The Mac-
Millan Co., 1920, Vol. 2, p. 578.
Marsh: Brit. M. J. 1:1356, 1909.
Sydenham: Brit. M. J. 1:1113, 1909.
Ney: "Facial Paralysis and the Surgical Repair of the Facial Nerve."
Laryngoscope 32:327, 1922.
Martin, R. C.: "Intratemporal Suture of Facial Nerve." Arch. Oto-
laryngology, 13:259, Feb., 1931.

VIII.

NASAL SINUS INFECTION ASSOCIATED WITH DISEASES OF THE VASCULAR SYSTEM.

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From the time that Virchow offered his theory of the inflammatory origin of atherosclerosis there have been repeated references to infection as a cause of cardiovascular disease. Ophüls[1] enumerates the various theories up to his time, and his statistical study appears to point to septic diseases as a factor.

Benson, Smith and Semenov[2] have reported the case of a girl, 14 years of age, with gangrenous stomatitis and fatal septicemia who presented the typical picture of beginning arteriosclerosis. Cultures from the mouth before death and of the blood at autopsy yielded a mixture of streptococcus hemolyticus and staphylococcus aureus. Autopsy revealed septic infarcts of the heart and lungs, disseminated acute myocarditis, thrombi in the right auricle and ventricle, and fresh patchy fatty degeneration of the aorta and larger arteries with beginning arteriosclerosis. Using cultures of streptococci obtained from sinuses and other sources, they gave repeated inoculations of broth cultures to a series of rabbits. Quoting their summary, they "described lesions in the aorta in rabbits inoculated with streptococci from sclerotic coronary arteries, coronary thrombi and sinusitis. These lesions include acute arteritis, with or without thrombosis and arteriosclerosis of the aorta and other arteries. The experimental changes are comparable with the corresponding arterial diseases found in man."

Langcope[3] and Libman[4] have maintained that thrombosis can occur with or without accompanying or preceding sclerotic changes in the vessel wall. Medical writing contains several ref-

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erences, and could contain more, of thrombosis arising suddenly in vessels which are not sclerotic.

In September, 1931, a woman, 54 years of age, was under my care for acute purulent sinusitis. She was in bed for two or three days with fever and pain over both maxillary antra. While convalescent and able to attend to part of her household duties, she was seized one morning with an attack of precordial pain. When seen by the internist a few hours later she had an ashy pallor, a rapid heart with very feeble heart tones. She had a low blood pressure and a temperature of 101. After thirty-six hours a slight precordial friction could be heard. Electrocardiogram made during the first thirty-six hours showed an inverted T wave in the first lead. After a few days' improvement she had a second attack of exactly the same sort with very feeble tones and gallop rhythm. There was fever off and on for two and one-half or three weeks. She was in bed for two months, making a gradual improvement. Since her convalescence she has gradually returned to her normal activities. She had no previous history of cardiovascular disease and there is now no physical evidence of such.

Inquiring among internists, I have been informed of three other cases with exactly the same history occurring in Portland. Considering the fact that the pain from coronary disease is sometimes referred to the upper abdomen, one wonders if some of the cases of so-called gastro-intestinal influenza with prolonged convalescence do not belong in the same category.

In our studies of the pathology of sinusitis at the University of Oregon Medical School we were impressed with the vascular and perivascular changes in the membranes from chronically infected sinuses; changes which often suggested those seen in syphilis. Semenov says: "In some cases of chronic sinusitis the arterial walls show some definite arteriosclerotic tendencies in young subjects. This may have some relation to the conception of infectious arteriosclerosis." While it would be natural that the vessels should partake of the inflammatory reaction of the tissues in which they are, Eggston[5] from his studies is inclined to believe that these vascular changes are an outstanding characteristic of sinus infection. He says: "The changes of paramount

value in classification have been of a vascular nature; the tissues in question have a circulatory system unique in structure and physiology. The lesions observed in the tissues from sinuses have been so universal that an attempt to explain the changes in the adjacent structures has been made with the vascular system as a basis." Speaking of the infiltration and edema, he says: "These reactions are primarily in the walls of the vessels and perivascular spaces and secondarily in the soft tissues. If the chief change in acute inflammation is vascular, it logically follows that the greatest chronic changes are also of a vascular nature." One of his suggested classifications is an "atrophic, fibrotic or arteriosclerotic" type. In his description of this stage or type of chronic sinusitis he says: "The most striking changes occur in and around the afferent blood vessels. Usually endarteritis, arteritis or periarteritis can be demonstrated. The vessel walls are often distinctly thickened. The lumen of the vessels is decreased and in some instances arteriosclerotic changes with complete occlusion of the vessels occur." These findings are in exact conformity with our own. In conclusion, Eggston remarks: "Perivascular reactions in acute inflammation are undoubtedly defense reactions. In the chronic processes there is a continuation of perivascular irritation by toxins, which accounts for the perivascular infiltration resulting in continued injury with fibrosis."

Repeated acute infections or the indefinite persistence of pathogenic organism in an attenuated form, in these tissues, would be logical cause of these progressive changes. We present the following as evidence of the presence of one of the factors.

Fresh tissues, removed at operation from chronic infected sinuses, washed, ground and cultured under aseptic precautions have given a predominating growth of streptococci in over 80 per cent of instances, and streptococci were present in over 90 per cent. These results were from a series of over 400 sinuses, mostly chronic nonsuppurating sinuses. The strains of streptococci in order of their frequency were: hemolytic B, green producing or hemolytic A, streptococcus viridans, and a nonhemolytic streptococcus of the gamma type. In combination with these were found micrococcus catarrhalis, pneumococci, Friedlander's bacilli, influenza bacilli, colon bacilli, diphtheroids, and streptothrix.

Staining for organisms in the tissues removed from sinuses, we have found consistently a diplococcus having the staining characteristics of the streptococcus. Occasionally other organisms were found conforming to the results from cultures. These organisms were most numerous and found most frequently in the deeper layers of the membranes, and were seen in the thickened walls of blood vessels.

With the idea that there might be a selective action for the blood vessels on the part of the organisms, we made a systematic search for organisms in blood vessel walls in sinus tissues removed from individuals having cardiovascular disease. So far we have sectioned the sinus tissues from eighteen such cases, in eleven of which we have found the organisms in the thickened vessel walls and accompanied by cell infiltration characteristic of chronic inflammation.

Turning to postmortem evidence, we have stained organisms in the walls of the coronary vessels from all of twelve cases showing definite cardiovascular disease. Most of the deaths were due to coronary occlusion. A few died from intercurrent disease. Ten showed fresh formed thrombi; two had the evidence of old thromboses. Cellular infiltration was present in all but two, and the bacteria were most numerous in the areas of infiltration. In sinus tissues the organisms were usually found in the adventitia and subintimal tissues. In the hearts they were most numerous about the vaso vasorum. The sections were cut with a thickness of five micros, and organisms were seen in one out of three of four sections. In a few they were found in all sections. They were not simply a casual occurrence.

We have then the theory of infection as a factor in the production of vascular disease.

The experimental production of vascular lesions by inoculations of pathogenic bacteria.

The presumptive clinical evidence that acute infection may produce vascular disease.

The histologic evidence of vascular changes being an outstanding incident in the tissues of infected sinuses.

The presence of morphologically identical organisms in the walls of vessels of the sinuses and of the heart.

These observations are presented in the spirit of investigation and not with the idea that any definite conclusions can be drawn from them.

MAYER BLDG.

BIBLIOGRAPHY.

1. Ophüls, W.: Arteriosclerosis: Cardiovascular Disease, Their Relation to Infectious Diseases. Stanford Univ. Pub., 1:1, 1921.

2. Benson, Smith and Semenov: Archives of Pathology, Vol. 12, December, 1931.

3. Longcope, W. T.: Jour. Iowa State Med. Soc., 12:314, August, 1922.

4. Libman, E.: Am. Heart Journal, 1:121, October, 1925.

5. Eggston, Andrew A.: Trans. Amer. Laryngological Assn., 1930.

6. Kistner, Frank B.: Archives of Otolaryngology, Vol. 13, February, 1931.

IX.

ABDUCENS PARALYSIS COMPLICATING OTITIS MEDIA AND MASTOIDITIS.*

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SANTA BARBARA.

Paralysis of the sixth cranial nerve occurring in the course of middle ear and mastoid infection has been the subject of numerous articles in the past few years. In the spring of 1933 three cases came under my observation during one month, and this was the incentive to the writing of this paper. A fourth case, which was treated in 1926, will be included in this report. The Gradenigo syndrome is not uncommon, but the number of cases occurring in the practice of any one otologist is quite limited. When we realize that[19] almost 20 per cent of the cases end fatally we are justified in viewing with alarm the occurrence of what is often considered a symptom of minor importance. Many otologists have completed their postgraduate training without having seen a patient presenting this syndrome. To such an otologist who sees his first case of sixth nerve paralysis complicating a middle ear and mastoid infection I would refer to the articles by Sears,[19] Eagleton,[35][36] Kopetzky and Almour.[1][3][4]

In 1904 Gradenigo described a symptom complex now known as the Gradenigo syndrome, which consists of (1) Acute otitis media or an exacerbation of a chronic otitis media with or without mastoiditis; (2) pain in the frontal-temporal-parietal region, and (3) a paralysis or paresis of the sixth cranial nerve on the diseased side appearing about a month after the onset of the otitis. Gradenigo regarded the frontal-parietal pain as an important part of the syndrome; but recently more emphasis has been placed on the sixth nerve paralysis. The term Gradenigo's syndrome is sometimes applied to all sixth nerve paralyses occurring in the

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course of middle ear and mastoid infection. Baldenweck and Prades[15] maintain that this is not correct, and many other authors agree with them. In three of the cases here reported the entire symptom complex was present. In the fourth case only a sixth nerve paralysis was found complicating a middle ear suppuration.

It is of more interest to note here that during the time the above three cases were under treatment, Dr. W. F. Mosher[37] of Ventura operated upon two cases of mastoiditis with abducens paralysis. Five cases were seen in a relatively small community and all occurred within a few weeks. This brings to mind the theory of specificity of organisms and its possible application to the involvement of the structures at the petrous apex.

A study of this condition necessitates mention of the structures about the petrous pyramid which may be affected by the infection. The cavernous sinus beginning anteriorly as a continuation of the ophthalmic vein and ending posteriorly in the superior and inferior petrosal sinuses. External to the cavernous sinus the two layers of the dura enclose a space over the apex of the petrous pyramid and containing the Gasserian ganglion. On the posterior surface of the pyramid, this dural separation encloses over the aqueductus cochlea and the saccus endolymphaticus. The carotid artery surrounded by a plexus of veins and passing through the carotid canal to enter the cranial cavity through the lacerated foramen. Branches of basilar must be mentioned here because of the close relation they sometimes have with the abducens nerve in its course between the clivus and the pons. Cushing,[2] in a study of abducens paralysis occurring with brain tumor, gives an illuminating picture of the relation of the branches of the basilar artery to this nerve. He demonstrates the possibilities of sixth nerve palsies being due to the constricting influence of these vessels.

The sixth nerve originates superficially in the groove at the junction of the medulla and lower border of the pons, a little lateral to the pyramid. It passes upward and lateral on the ventral surface of the pons for about 15 mm. and pierces the dura a little above the junction of the basilar process of the sphenoid with the occipital. It turns forward in the interval between the apex of the petrous and the posterior clinoid process of the

sphenoid under a ligament, which connects the bony processes mentioned. This briefly describes the formation of the so-called Dorello canal through which the sixth nerve passes and at which point strangulation of the nerve occurs. Glick,[34] in a number of specimens, has observed bony dehiscences directly under the Gasserian ganglion, and in many specimens only a very thin layer of compact bone separated the ganglion from the cellular spaces. He concludes that pneumatic cells are present in the petrous apex more frequently than is generally supposed. Wolff[23] describes apertures occurring in the bony wall of the carotid and facial canals. Profant[27] made dissections of the temporal bones of fifty adults, and concludes that pneumatic cells are present in the petrous bone more frequently than is generally known, and suggests the name "petrositis" for infection of these cells. He describes two possible routes of entrance for the infection, (1) the antrum-epitympanic, and (2) the hypotympanic. Other observers have mentioned three or four routes of infection, but it seems quite possible that the extension could take place through any contiguous group of cells, and in this way any number of routes could be followed.

Eagleton[35] has made investigations to determine the various anatomic factors concerned in the production of the facial pain and abducens paralysis in suppuration of the petrous and meninges. Also to determine why the symptoms developed in one case and not in another. Applying the knowledge gained so that we may recognize the cases in which intracranial complications are most likely to develop and institute early treatment. It is only in the primary stage of the meningeal process that surgical intervention promises any measure of success. He concludes that facial pain in suppurative disease is diagnostic of middle fossa involvement and that posterior fossa involvement does not cause facial pain. All cases with facial pain that disappears after a simple mastoid operation are probably due to irritation of one of the sensory communications of the fifth and not the trifacial nerve. In caries of the petrous apex the pain is due to irritation of the semilunar ganglion or its first branch, because of dural adhesions, and this type of pain is not likely to be influenced by mastoid operation. He concludes that on anatomic grounds and from clini-

cal experience, pain back of the eye may be the first manifestation of congestion or granulation caries of the petrous apex. Anatomic variations have much influence on the liability to abducens paralysis in different patients. The length of the nerve, its position on bony basis, the extent of closure between the petrous apex and the occipital and sphenoidal base, the closeness of its attachment to periosteum, and lastly the great variation in the size of Dorello's canal. He reports two cases of caries of the petrous apex from suppurative otitis presenting the suggestive symptom of "pain behind the eye." Autopsy revealed large carious cavity involving the apex and extending to both anterior and posterior surfaces of the pyramid.

Vail[6] made studies to determine the source of the typical pain in petrous apex suppuration. He felt that it was not due to irritation of the ophthalmic division of the fifth, but was produced by the irritation of the great superficial petrosal nerve caused by the suppuration in the petrous bone.

Geyman and Clark[21] describe their method of roentgen demonstration of suppuration in the petrous pyramid. They use two positions, (1) fronto-occipital (Fig. 1) and (2) vertico-mental (Fig. 2). They do not feel that a positive diagnosis should be made on roentgen findings alone, but it has a definite value in affirming or denying a clinical diagnosis of petrous suppuration.

On several occasions the writer has noted improvement in mastoid symptoms after exposure to roentgen ray for a series of plates. The improvement may have been coincidental and it probably was. In discussing with roentgenologists the therapeutic value of X-rays in mastoid infection, they state that little, if any, effect could be expected. Van Voorthuysen[17] reports one case of Gradenigo's syndrome in a child 9 years of age, in which the headache, abducens palsy and symptoms of labyrinthitis remained unchanged for several weeks after an acute otitis, but suddenly disappeared two days after exposure to the X-rays for a series of radiograms. Gradenigo syndrome is rarely associated with temporosphenoidal abscess, but Roberts[16] reports a case in a boy, 16 years of age, who developed an acute suppurative otitis media on the left side, after influenza. The otitis was com-
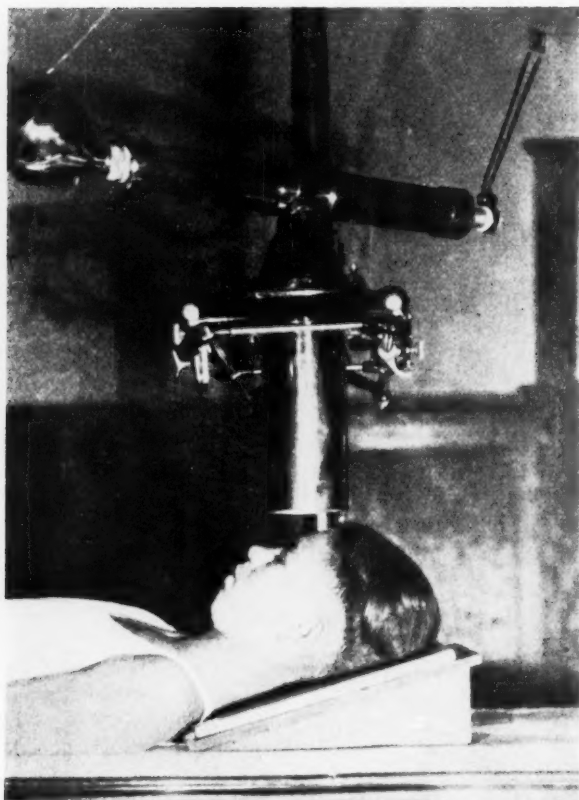
Fig. 1.   Fronto-Occipital Position, Geyman and Clark.[21]

plicated by severe temporal pain on the same side and paralysis
of the sixth nerve. Subsequently a temporosphenoidal abscess
developed which proved fatal. The author notes that Gradenigo's
syndrome is rarely associated with abscess.

Baldenweck and Prades[15] report a typical case in which a
complete paralysis of the sixth nerve on the left side, preceded and
accompanied by severe pain in the frontal-parietal-temporal re-
gion, developed five weeks after the onset of an acute otitis in
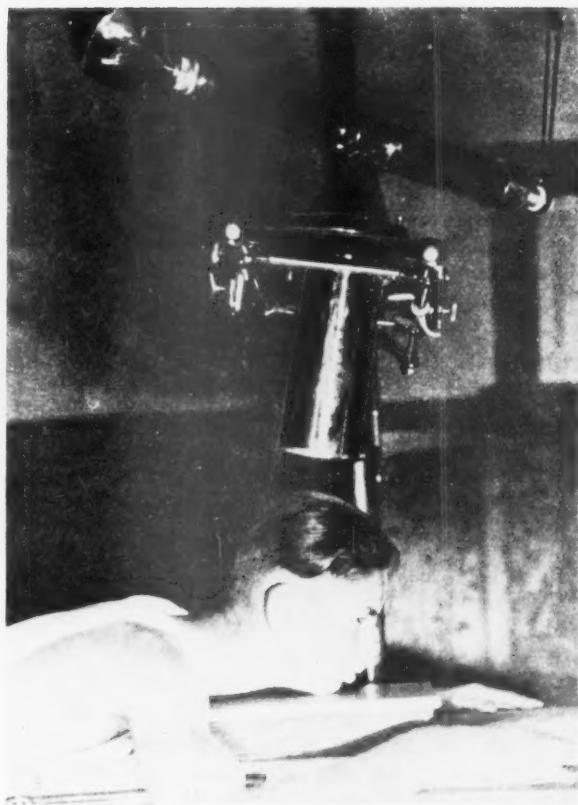
Fig. 2.   Vertico-Mental Position, Geyman and Clark.[21]

the left ear and ten days after the mastoid operation. Fifteen days after the appearance of the sixth nerve paralysis, the patient had severe vertigo and vomiting, spontaneous nystagmus to the left, no signs of meningitis or cerebellar lesion. Radiographic studies made in this case showed the point of the petrous bone to be entirely obscured, indicating the presence of an osteitis with purulent resorption. The remainder of the petrous bone showed an abnormal degree of transparency, evidently due to rarefaction

resulting from the purulent osteitis of the tip. This osteitis of
the tip of the petrous bone evidently caused the labyrinthine symp-
toms noted, as well as the sixth nerve paralysis. The symptoms
subsided gradually with only a slight residual paresis of the sixth
nerve three months later.

Boonacker and Huizinga[14] report seven cases in which abdu-
cens paralysis occurred as a complication of otitis media. In two
of these cases the otitis was catarrhal and there was no mas-
toiditis; in the other five cases the otitis was of the acute sup-
purative type, but in one of these in which operation was done
because of the sixth nerve paralysis no mastoid infection was
found. Five of the cases showed the severe pain in the fronto-
temporal-parietal region described by Gradenigo; one had only
a slight headache; and one no symptom in addition to those of
otitis and mastoiditis except the abducens paralysis. In none of
these cases was there any evidence of intracranial infection, men-
ingitis or labyrinthitis. All the patients recovered, and the sixth
nerve paralysis disappeared entirely. Six of these cases occurred
during an epidemic of influenza and the authors are inclined to
believe that the neurologic symptoms in these may have been
due to influenzal infection rather than a direct complication of
the otitis.

Greenfield[13] reports a case of acute otitis media and mas-
toiditis in a child, 6 years of age, in which severe pain—parieto-
temporal and retrobulbar—and the abducens paralysis did not
develop until a month after a simple mastoidectomy had been
done. The pain was chiefly retrobulbar. The mastoid wound
was practically healed when the abducens paralysis developed.
The patient made a gradual recovery, the abducens paralysis
having nearly disappeared on discharge from the hospital. The
author notes that it is the consensus of opinion that in most cases
of otitis in which abducens paralysis occurs there are definite
evidences of involvement of the mastoid. As a rule, the mas-
toiditis is of a severe type and the necrosis extensive. In the
author's case this was true; the zygoma was entirely broken
down. The appearance of the Gradenigo's syndrome (pain and
sixth nerve palsy) varies in relation to the course of the otitis.
It may develop during the middle ear stage, at the height of the

mastoid infection, or only after the mastoid operation—even some weeks after, as in the author's case. In all the cases a profuse aural discharge persists.

Jungert[12] notes that not every case of paralysis and paresis of the abducens (sixth cranial) following acute otitis shows the typical Gradenigo syndrome. Paralysis of the sixth nerve may be associated with paralysis of other cranial nerves in diffuse suppurative meningitis and brain abscess of otitic origin. And Gradenigo has noted that in cases showing his syndrome, other nerves may also be involved (trigeminal, oculomotor), which apparently stand in no etiologic relationship to the abducens paralysis. The Gradenigo syndrome may also be complicated by diffuse suppurative meningitis, terminating fatally.

At the Sabbatsberg Clinic,[12] nine casets of the Gradenigo syndrome have been observed since 1918; seven of the patients were under forty years of age; in all the onset of the abducens paralysis was sudden; in six cases the abducens paralysis occurred thirty to fifty days after the onset of the otitis. In five cases it occurred after the mastoid operation had been done. In most cases it disappeared gradually, often within a month. In one case the abducens paralysis was bilateral and there was pupillary stasis, but in this case there was a complicating thrombosis of the internal jugular vein and the sigmoid sinus. Except in this case, in which there was also pyemia, the temperature was either normal or subfebrile. Of the five cases in which the abducens paralysis occurred after the mastoid operation it subsided without further operation in one case. The other four cases were re-operated and in two of these an abscess was found in the tip of the petrous bone. In the four cases in which a mastoid operation was done after the development of the abducens paralysis, the radical operation was done in one case, and in this case an abscess was also found in the tip of the petrous. In two of these cases roentgen ray examination had shown the tip of the petrous bone to be destroyed. These cases support Gradenigo's opinion that a localized osteitis of the tip of the petrous is the cause of the abducens paralysis in cases showing his syndrome. The author is of the opinion that in cases of Gradenigo's syndrome in which roentgenologic examination shows changes in the tip of the petrous bone, even the radical

mastoid operation is not sufficient, but the operative field must be extended in order to drain this portion of the petrous bone.

Meltzer[11] maintains that a circumscribed osteitis of the tip of the petrous bone eventually producing a corresponding localized leptomeningitis involving the abducens nerve and Gasserian ganglion by contiguity, is not necessarily the only explanation of the typical pain and sixth nerve paralysis of the Gradenigo syndrome. The sixth nerve, he notes, has but a small area of contact with the petrous tip. He concludes that it is reasonable to suppose, in the light of recent studies, that the Gradenigo syndrome may be due to: "(a) cells extending to the apex (paralabyrinthine subarcuate, paratubal) ; (b) the carotid canal . . . . by erosion of the bone wall of the eustachian tube ; (c) the perineural and perivascular lymphatics of Papule ; (d) erosion of the tegmen tympani with extension forward, and (e) the inferior and superior petrosal sinus."

Frenckner[30] reports two cases of Gradenigo's syndrome following acute otitis, in one of which there was also a paresis of the facial nerve. The X-rays showed a destructive process in the apex of the petrous bone in each case. The author operated upon both these cases with drainage of the petrous apex. Both patients made a good recovery, and later X-ray studies showed increased calcium deposition and regeneration of bone in the apex. In his operation on such cases the author does either a thorough mastoidectomy or a radical mastoid operation, with very thorough evacuation of the cell system; the three semicircular canals are uncovered ; a fine meshed cell system can usually be seen entering the upper canal. A small scoop or curette is used to follow these cells inward and thoroughly scrape the convexity of the canal ; the curette is advanced to the apex, and any pus, necrotic or hemorrhagic tissue or granulations scraped away. When a cavity with firm walls is found, this is drained with a fine rubber membrane, changed daily. A small scoop is used to clear the entrance of the semicircular canal and the area nearest the cavity to prevent accumulation of waste matter.

Burger[29] reports two cases in children in which unilateral paralysis of the sixth nerve developed following acute otitis and mastoiditis, after the mastoid operation had been done. In one

case there was no other symptom; in the other there was also a temporary facial paralysis and symptoms of meningeal involvement. In the first case the abducens paralysis subsided without further treatment; in the second case a radical mastoid was done and the child made a good recovery. In both these cases roentgen ray examination made after the abducens paralysis had occurred showed a destructive process in the apex of the petrous bone; in the first case the defect was extensive—no trace of bone tissue being demonstrable in the apex. A year later another roentgenogram showed marked bone regeneration.

Malan[28] reports a case in a man, 58 years of age, in which a paralysis of the sixth nerve on the left side followed an acute otitis on the same side. The symptoms of mastoiditis were slight, but mastoidectomy showed widespread involvement of mastoid cells. After the operation the Gradenigo pains in the temporal and retrobulbar region developed. The X-ray showed a decalcifying destructive process in the left petrous bone. After a second operation the sixth nerve on the right side and pain on the right side of the head developed, with fever and signs of meningitis. The patient died and autopsy showed suppurative meningitis at the base and diffuse osteitis of the petrous bone on the left side. Two cases are reported by Profant.[27] In the first of these cases, pain in the temporoparietal region developed on the twenty-fifth day—the twelfth and fifteenth day after the mastoid operation. Subsequently generalized meningitis developed that proved fatal.

In the second case severe pain developed on the tenth day of the otitis; the paralysis of the abducens, on the thirteenth day. Mastoidectomy with wide exposure was done and drained through the antrum—epitympanic route. There was generalized meningitis with death on the fifteenth day.

From his[27] study of these cases and a review of nineteen cases from literature (since 1924), the author finds that the typical pain and abducens paralysis of Gradenigo's syndrome may appear with no manifestations of mastoiditis, or before, during or after mastoiditis. The majority of patients are relieved of symptoms by a simple mastoidectomy. Cases in which the syndrome develops after mastoidectomy are "more alarming." The pain is usu-

ally temporoparietal, but may involve all branches of the trigemi-
nus; it usually precedes the paralysis of the sixth nerve.

Freud[26] reports a case in a boy, 9 years of age, in which abdu-
cens paralysis developed following the mastoid operation. In
this case the paralysis of the abducens preceded the onset of the
unilateral facial pains characteristic of the syndrome. Another
unusual feature was a transitory ptosis and edema of the upper
eyelid on the opposite side with facial neuralgia on that side. The
patient was given a blood transfusion and recovered without fur-
ther operation. In a review of Gradenigo's syndrome, Freud em-
phasizes the following points: This syndrome occurs in children
oftener than in adults; the sequence of symptoms is not always
the same; it usually occurs three to four weeks after the onset of
an acute otitis. At times an unexplained complication will occur,
as in the author's case. In treatment a Schwartze mastoidectomy
usually is sufficient.

Adelstein[9] reports two cases presenting the syndrome but no
definite evidence of mastoiditis. Operation revealed infection in
the mastoid of both patients and both recovered. He is of the
opinion that the occurrence of a sixth nerve paralysis and pain in
the eye or over the frontotemporal region on the same side as an
acute otitis "calls for" an investigation of the mastoid and mas-
toidectomy even if the X-ray picture is inconclusive. In cases
with no evidence of mastoid involvement, the extension of the
infection from the middle ear to the petrous tip may be (1) from
the tympanum below the labyrinth and internal auditory meatus
to the petrous tip; (2) by way of the carotid canal by erosion of
the bone, or of the tympanic foramina; or (3) by suppuration fol-
lowing through a layer of cells extending along the eustachian
tube from the middle ear to the petrous portion of the mastoid.

Trivas and Trixier[24] report a case of a 15-year-old boy, in
which in the fourth week of a suppurative otitis, a paralysis of
the sixth nerve and severe frontal and temporal pain developed on
the same side as the otitis. There was some fever and slight signs
of meningitis. The mastoid was tender to pressure, but there
were no other symptoms of meningitis. At operation an extensive
involvement of the mastoid was found. After operation the sixth

nerve paralysis subsided rapidly, but symptoms of serous meningitis persisted and even increased for a time. The patient, however, made a good recovery.

Bigler[10] reports a case in a girl, 12 years of age, in whom a suppurative otitis developed after an attack of influenza. A roentgen ray examination showed pneumatization of the mastoid on both sides; on the side with no ear infection pneumatization of the petrous was also demonstrable, but on the diseased side there was a diffuse shadow obscuring the upper portion of the petrous. As the neuralgia-like pain precedes the abducens paralysis as a rule in the Gradenigo syndrome, Bigler is of the opinion that roentgen ray examination made at the time of the appearance of such pain in cases of otitis would reveal the infection of the apex of the petrous before the development of the sixth nerve paralysis.

Veits[25] reports two cases of suppurative otitis with intracranial complications in which abducens paralysis occurred on the side opposite to the discharging ear. In one the sixth nerve paralysis was associated with extensive thrombosis of the cavernous sinus, and in the other with a temporal lobe abscess (on the same side as the otitis). In this latter case the author considers that the abducens paralysis was due to increased intracranial pressure and strangulation of the abducens by the branches of the arteria basilaris as described by Cushing. In cases of sinus thrombosis, abducens paralysis on the opposite side may be due to asymmetry of Dorello's canal, rendering the nerve on the opposite side more liable to injury from edema developing suddenly. Veits notes that abducens paralysis on the side opposite to the otitis is of very rare occurrence.

Guthrie[32] reports a case with contralateral paresis of abducens and double optic neuritis following operation. The complications in this case were probably due to obliteration of the lateral sinus after accidental injury at the mastoid operation. The patient recovered.

Hall[33] reports a case almost identical with Guthrie's, except that the jugular was ligated and the sinus obliterated for thrombosis. Papilledema developed on the third postoperative day and abducens paralysis on the nineteenth. This patient recovered.

Berens[8] notes that involvement of the sixth nerve is the most common motor disturbance of the eye accompanying diseases of the ear. "The sixth nerve is attached to the pons and more or less held in the cavernous sinus. It will therefore be pressed against the sharp border of the petrous temporal bone by any condition which causes downward pressure on the brain, with resulting interruption of conduction and palsy of the external rectus muscle."

Ryland and Girling[7] report a case of masoiditis, localized purulent meningitis with purulent cerebrospinal fluid and paralysis of sixth and seventh. No improvement following simple mastoid but recovery after a radical. Bacteria found in cerebrospinal fluid but no chemical or cultural characters of the fluid were given.

Seydell[20] reports two cases, one of which developed a lateral pharyngeal abscess similar to that described by Eagleton. Seydell describes a method of draining the tip cells by going through the groups of cells which lie anterior or posterior to the capsule of the labyrinth. The technic employed does not require special apparatus or instrments, it is more simple and requires less time than many of the other operations described.

Simpson[31] reports a series of three cases and concludes that in some cases of otitis showing Gradenigo's syndrome there is very little clinical evidence of mastoiditis, but the author has noted in studying his own cases and reports of other cases that there is usually quite a definite mastoiditis of a severe type, and also that there is a tendency for a discharge through the external auditory meatus to persist although the mastoid wound heals quickly. In cases showing this syndrome the author believes an early mastoid operation is indicated—a little more thorough than usual "in hunting for infection, such as in the zygomatic cells, etc."

Whitman[18] reports a case in which the pain and sixth nerve paralysis did not occur until after a simple mastoidectomy had been done; just previous to this there had been a recurrence of pain in and discharge from the ear. A secondary simple mastoidectomy was done with exploration of the middle fossa; the zygoma and region of the posterior semicircular canal were cleaned out as far as possible. The entire petrous bone appeared

to be pneumatic. Pain was relieved in a week after operation, and the sixth nerve paralysis cleared up gradually in two months.

Bernstein[5] reports a case in a boy, 10 years of age, in which a suppurative otitis in the left ear was accompanied by headache and sixth nerve paralysis on the same side. There was no mastoid tenderness and no fever. A simple mastoid operation was done; extensive pneumatization of the mastoid process was found, and antrum and cells filled with pus under pressure and granulation tissue. The headache subsided and the sixth nerve paralysis cleared up in three days. But in ten days the sixth nerve paralysis and headache recurred, accompanied by vomiting and slight cervical rigidity. The temperature and pulse rate rose, the vomiting became cerebral in type and a "naming" aphasia developed, indicating the development of a temporosphenoidal abscess. A radical mastoid operation was done, the abscess found and drained, and the patient made a good recovery.

Kopetzy and Almour[1,3,4] conclude that petrous apex involvement takes two forms: Acute, threatening meningeal involvement, or chronic, which does not threaten meningeal involvement. That the petrous pyramid is a complication of tympanic and not mastoid infection. From their results they believe that the technic they advocate establishes adequate drainage in cases of petrous pyramid suppuration. Eagleton[36] describes his operation of unlocking the petrous pyramid in the treatment of localized bulbar meningitis secondary to petrous apex suppuration and reports four cases operated upon, with three recoveries.

The presence of this syndrome may justify surgical interference but we must admit that the procedure is a difficult one to carry out. We must decide which is to the best interests of the patient: (1) A conservative course with the dangers of a possible meningitis, and (2) operative interference with the added risk of injury to the structures located in the petrous pyramid. The problem then, in which cases shall we attempt to drain the petrous apex and when, is always a difficult one to decide.

Case 1.—J. L., female, age 11, was first seen on March 24, 1933, complaining of pain in the right ear; she had contracted quite a severe cold a few days previous. The upper part of the tympanic membrane was red but there was no bulging. The next day there was a definite bulging and

myringotomy was performed. The left ear was normal. The discharge was very profuse and warm saline irrigations were used every few hours. There was slight tenderness on pressure on mastoid twenty-four hours after myringotomy. It was impossible to get this child to take even an ordinary amount of fluids and she refused almost all food. During the first week temperature ranged from 99.5 to 103. She vomited quite often and taking food would usually bring on vomiting. Seven days after the opening of tympanic membrane the urine had a smoky appearance and when examined a large amount of blood with casts was reported. The white cell count was 17,000 with 88 per cent polynuclears. Cultures from the ear showed strepcocucus viridans. There was a gradual increase in the edema on the mastoid process.

April 3rd the X-ray laboratory reported symmetrical cell development on both sides. Left mastoid normal. On the right side there is a complete loss of air and a generalized cloudiness involving the entire mastoid area. In the posterior portion there is evidence of disintegration of cell septa. The X-ray did not include the petrous pyramids. The discharge from the ear continued to become even more profuse than at the beginning of the week.

April 4th, a right simple mastoid operation. Very extensive pneumatization extending well posterior to the sinus and up into the zygoma. Some breaking down of septa, but no dura or sinus wall exposed. Gauze drain was inserted into antrum and upper part of incision closed with silk worm gut sutures. April 5th, the temperature was 102.4 and the patient was quite comfortable.

April 9th removed sutures and drains from ear canal and mastoid cavity. Much more pus than usually seen. Kidney condition unchanged; temperature 101.4 in evening. April 12th, the eighth postoperative day, she complained of pain in side of head, some photophobia and vomiting. She also complained of double vision and on examination it was found that she had a complete paralysis of the sixth on the right side. During the next eight days the temperature ranged from normal to 101, the white cell count 11.000 to 23.000 and the discharge from ear and mastoid was profuse; sixth nerve still paralyzed. Urine contained blood. She was discharged from the hospital on April 20th after the temperature had remained below 99° for two days.

The ear and mastoid were dressed daily in the home, drainage decreased and wound almost healed after ten days. The urine still contained blood and it was impossible for her to retain fluids. She complained of no pain in head. The eyegrounds were normal on repeated examinations in hospital and in the home. April 30th she seemed to be much improved and moved about the bed and sat up most of the day. In the evening she had considerable pain in the head, some neck rigidity, positive Kernig and the temperature was 103.5. The urine was scanty and contained blood.

May 5th she re-entered the hospital. During the next few days the meningeal symptoms cleared up but vomiting and blood in urine continued. Further surgical treatment was not advised on account of her poor general condition. Fluids by rectum, intravenous, and blood transfusions were of no avail and she expired on May 15. Unfortunately it was impossible to obtain a postmortem.
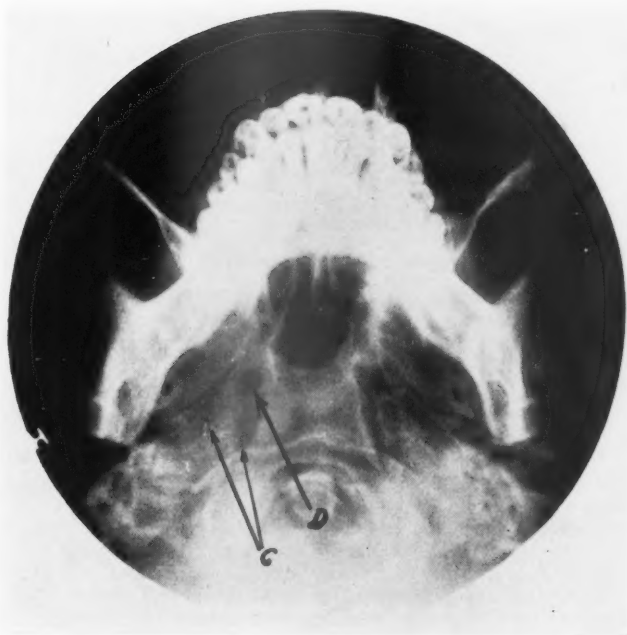
Fig. 3. Vertico-Mental position Case 2. *C* diseased area of petrous,
D Dorello's canal.

Case 2.—A. A., female, age 13, first seen on March 31, 1933. History
of acute upper respiratory infection for five days and pain in the right
ear for three days.

The left tympanic membrane was normal, the right was red and bulging
and was incised. She complained of much pain in the ear and entire side
of the head. The eyegrounds were normal. The discharge from the ear
was not profuse; temperature normal to 100. White cell count 16,400.
April 5th complained of double vision and it was found that she had
paralysis of the right abducens (Fig. 4). X-ray studies show normal left
mastoid cloudiness of right but no disintegration of septa. Studies of the
petrous pyramids (Fig. 3) in the verticomental and Towne positions show
a normal left and definite displacement of air and a cloudiness of tip
cells on right. Diagnosis, (1) late first stage mastoiditis right, (2) acute
right petrositis.

April 6th. Pain in head quite severe; some mastoid tenderness. Eye-
ground examination shows bilateral papilledema. There was no blurring
vision. R., V. 20/15; L., V. 20/15.

Fig. 4. Case 2. Looking to her right, showing paralysis of right abducens.

April 10th. Check-up X-ray studies showed very little change in past five days.

April 17th. X-ray studies showed advancement in the process and disintegration of septa. Eyegrounds show no change; vision 20/15 in each eye. Temperature ranges from normal to 99.5. Some mastoid tenderness and sagging of posterior superior canal wall. No edema on mastoid process. White cell count 10.000. Urine normal.

April 19th. Right, simple mastoid operation. Small type cells, all filled with pus and granulations; much disintegration of septa throughout. The anterior bony wall of the sinus from just below the knee to the bulb necrosed and the membranous sinus wall was covered with granulations. No dura exposed in other regions. The cells extended well up into zygoma, but no definite route could be determined as to the petrous pyramid infection. Bacteriologic examination from mastoid showed streptococcus viridans. Patient remained in hospital for 12 days and the convalescence was uneventful. The abducens paralysis began to improve on the fifth postoperative day, but it required about four weeks for the function to completely return (Fig. 5). The papilledema did not entirely disappear for about seven weeks, but the vision remained 20/15 in each eye.

September 2, 1933. Eyegrounds and fields are normal. Vision 20/15 each eye and normal hearing in the operated ear.

Fig. 5. Case 2. About four weeks later, looking to her right, showing return of abducens function.

Case 3.—V. S., female, age 13. First seen March 15, 1926. History of many attacks of middle ear infection and a mastoid on the left side. Pain in right ear for one day, tympanic membrane red and bulging. Incised and found middle ear filled with serosanguinous fluid. Six days later there was right abducens paralysis and pain in parietal and temporal region and some mastoid tenderness. X-ray studies showed a cloudiness of the right mastoid. On the 10th day a simple mastoid operation was performed. All cells were filled with pus and extended far up into zygoma. No dura or sinus was exposed. White cell count was 12,500. Urine examination negative, temperature range 99 to 100.5. Eyegrounds normal. Recovery was uneventful, and function of abducens had completely returned at the end of two weeks. Hearing returned to normal.

Case 4.—D. D., female, age 5½ years. First seen June 2, 1933. Parent gave history of the child having left earache two weeks ago and the tympanic membrane being incised. Pain in ear and head was severe but opening the ear gave relief. The mother states that the left eye turned in for three days before the ear drum was incised.

Examination. Left tympanic membrane healed, hearing normal, complete paralysis of left abducens, blurring of the disc in both eyegrounds and vision was normal as near as could be determined.

Two weeks later the abducens function had returned and the eyegrounds were normal.

CONCLUSIONS.

Forty-five cases have been considered in this report. There were eight deaths, or about 17 per cent. This is about the same rate as is given by Sears in his report of a review of more than 250 case reports.

There is a lack of reports of autopsy findings, but from the evidence to date we must conclude that petrous pyramid suppuration due to extension from the middle ear or mastoid is not a rare condition.

Patients with large pneumatic mastoids will probably have extensive cell formation in the petrous pyramids. Routine roentgen ray studies of the petrous pyramids in all cases of mastoiditis will give us valuable information.

Anatomic variations of the structures at the petrous apex are such that the typical syndrome is produced in only part of the cases developing localized bulbar cisterna meningitis.

The petrous pyramid route is probably the most common one in cases developing meningitis following mastoid infection.

Lastly I make a plea for more complete postmortem examinations, including the removal and microscopic study of the petrous pyramids in all cases dying of intracranial complications following middle ear and mastoid disease.

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BIBLIOGRAPHY.

1. Kopetzky, S. J., and Almour, R.: Suppuration of the Petrous Pyramid: Pathology, Symptomatology and Surgical Treatment. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. XXXIX, No. 4, Vol. 1, XL., Nos. 1-2-3, 1930-1931.

2. Cushing, H.: Strangulation of the Nervi Abducens by Lateral Branches of the Basilar Artery in Cases of Brain Tumor. Brain, 1910, XXXIII, 204-235.

3. Kopetzky, S. J., and Almour, Ralph: Empyema of the Petrous Apex. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY. Vol. XLII, No. 3, P. 828.

4. Almour, Ralph: Empyema of the Petrous Apex: Operation, Recovery. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. XLI, No. 2, 1932.

5. Bernstein, S: A Case of Temporosphenoidal Abscess Associated with Gradenigo's Syndrome. J. Laryng. and Otol., 48:196, March, 1933.

6. Vail, Harris N.: Virian Neuralgia. Archives of Otolaryngology, V. XVII, February, 1933.

7. Archer, Ryland, and Girling, W. N. M.: A Case of Localized Purulent Meningitis with Purulent Cerebrospinal Fliud in the Course of Acute Mastoiditis Occurring in a Highly Cellular Bone and Complicated by Paralysis of the Sixth and Seventh Cranial Nerves on the Affected Side. Journal of Laryngology and Otology, V. 44, 1929, P. 467.

8. Berens, C: Intracranial Lesions of Otitic Origin: The Ophthalmologist's Point of View. Trans. Amer. Otol. Soc., 21:67, 1931.

9. Adelstein, L. J.: Gradenigo's Syndrome and Brain Abscess. California and Western Med., 34:23, 1931.

10. Bigler, M.: Zur roentgenoligischen Darstellung eines Knochenabscesses in der Felsenbein Spitze Beim Gradenigoschen Symptomemkomlex. (Roentgenological Demonstration of a Bone Abscess in the Apex of the Petrous Bone in the Gradenigo Syndrome.) Ztsch. f. Hals-, Nasen- u. Ohrenh., 25:249, 1930.

11. Meltzer, P. E.: Gradenigo's Syndrome: Anatomic Aspects. Arch. Otolaryng., 13:87, 1931.

12. Jungert, Y.: Einiges uber das Syndroma Gradenigo und die Erfahrungen Daruber and der Klinik auf Sabbatsberg (Gradenigo's Syndrome with Cases from the Sabbatsberg Clinic). Acta Oto-Laryng., 11:394, 1931.

13. Greenfield, S. D.: Gradenigo's Syndrome. Arch. Otolaryng., 9:171, 1929.

14. Boonacker, A. A., and Huizinga, E.: Acta oto-laryn., 13:65, 1928.

15. Baldenweck, L., and de Prades, D.: Application de Radiographie du rocker a l'etude du syndrome de Gradenigo. (Radiography of the Petrous

Bone in the Study of Gradenigo's Syndrome). Acta oto-laryng., 11:244, 1927.

16. Roberts, E. R.: Acute Otitic Temporosphenoidal Abscess. Arch. Otolaryng., 6:420, 1927.

17. van Voorthuysen, D. G. W.: The Origin and Development of Gradenigo's Syndrome. (English text.) Acta oto-laryn., 18:265, 1933.

18. Whitham, J. D.: Gradenigo's Syndrome. Laryngoscope, 37:368, 1927.

19. Sears, W. H.: The Gradenigo Syndrome. Laryngoscope, 37:32, 1933.

20. Seydell, Ernest M.: Surgical Treatment of Suppuration of the Petrous Pyramid. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. XLII, No. 2, June, 1933.

21. Geyman, Milton J., and Clark, Daniel M.: The Roentgen Demonstration of Petrositis. Acta Radiologica, V. XIII, 2-72.

22. Berens, Conrad: Intracranial Lesions of Otitic Origin. Trans. American Otological Soc., 1931.

23. Wolf, Dorothy: Microscopic Observations of the Eustachian Tube. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 40, Dec., 1931.

24. Trivas and Trixier: Contribution l'etude du syndrome de Gradenigo. Rev. de laryng., 50:541, 1929.

25. Veits, C.: Zwei Falle von Seitengekreuzter Abduzensparese bei Endokraniell Komplizierter Otitis. (Two Cases of Abducens Paresis on the Opposite Side in Otitis with Intracranial Complications). Folia oto-laryng., Pt. 1, 20:447, 1931.

26. Freund, E. M.: Paralysis of the Abducens Nerve Accompanying a Case of Mastoiditis. Arch. Otolaryng., 13:717, 1931.

27. Profant, H. J.: Gradenigo's Syndrome. Arch. Otolaryng., 13:347, 1931.

28. Malan, A.: Syndrome de Gradenigo Bilateral. Ann. d'oto-laryng., No. 2:129, 1931.

29. Burger, H.: Die Roentgenologische Diagnose des Syndroms von Gradenigo. (Roentgenological Diagnoses of Gradenigo's Syndrome). Acta oto-laryng., 17:353, 1932.

30. Freckner, P.: Some Remarks on the Treatment of Apicitis (Petrositis), With or Without Gradenigo's Syndrome. Acta oto-laryng., 17:97, 1932.

31. Simpson, W. L.: Gradenigo's Syndrome. Gradenigo's Syndrome with Report of Case. Laryngoscope, 37, October, 1927.

32. Douglas, Guthrie: Acute Mastoiditis, With Sixth Nerve Paresis and Double Optic Atrophy. Journal of Laryngology and Otology, Vol. XLVIII, No. 12, p. 839.

33. Hall, I Simpson: Acute Mastoiditis, with Sixth Nerve Paresis and Double Optic Atrophy. Journal of Laryngology and Otology, Vol. XLVIII, No. 12, p. 840.

34. Glick, H. N.: Microscopic Observations of the Petrous Apex. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. XLII, No. 1, 1933.

35. Eagleton, Wells P.: Localized Bulbar Cisterna Meningitis, Facial Pain and Sixth Nerve Paralysis and Their Relation to Caries of the Petrous Apex. Archives of Surgery, Vol. XX, March, 1930.

36. Unlocking the Petrous Pyramid for Localized, Bulbar Meningitis Secondary to Suppuration of the Petrous Apex. Archives Otolaryngology, Vol. XIII, March, 1931.

37. Mosher, W. F.: Ventura, California. Personal communication.

X.

A CLINICAL AND EXPERIMENTAL STUDY OF THE ACTION OF SALIVA ON BLOOD COAGULATION AND WOUND HEALING IN SURGERY OF THE ORAL CAVITY AND THROAT.*

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For many years saliva has had a prominent place in medical literature. Despite the profusion of these writings, most of them deal with the physical and chemical properties of the saliva, little stress being laid on its potentialities as a factor in the process of coagulation and healing. There is, indeed, an amazing dearth of information of this phase of the subject. We have made an attempt to delve into the subject and enlarge the fund of knowledge concerning it.

A great deal may be learned from animals. Their functions, though simple in character, are effective in accomplishing their purposes. We know that the hurt animal will lick an injury back to health. Man, by instinct, puts an injured finger into his mouth.

From a laboratory standpoint, saliva is of little interest to the clinician, but when utilized for clinical purposes the subject assumes an entirely different aspect.

Volumes have been written on the subject of hemorrhages following operative procedures in the throat and oral cavity. Much has been said about the method of control, and the invaluable aid of clamps, sutures and various chemical and biologic coagulants. The salivary secretions, although they too have a specific function to perform in the control of hemorrhage, have been neglected. Our observations in vivo as well as in vitro have shown conclu-

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sively that saliva, mixed with blood, becomes a potent factor in decreasing the coagulation time.

A popular and oft expressed theory is that if oozing follows a tonsillectomy, it will be controlled by the patient's vomiting. Knowing that vomitus is acid in reaction, and prompted by curiosity, a drop of blood was placed upon a microscope slide, to which was added a drop of weak hydrochloric acid solution. It was found that the coagulation time was markedly increased. To another drop of blood was added a drop of saliva, and a marked decrease in the coagulation time was noted.

A review of the existent literature disclosed that the effect of saliva on the coagulation of the blood was mentioned by Grevin in 1918,[1] Hunter in 1928[2] and Bellis, Birnbaum and Scott in 1932.[3]

Grevin's writings were philosophical in their nature. He noted that animals healed their wounds by licking them and thought that "one day someone would find in one or several of the components of saliva something that would be of value in the treating and curing of many wounds, which were the despair of the wounded soldiers who presented themselves for surgical treatment."

His wish for further work in this field was not fulfilled until 1928, when Hunter also noted, from his observation of animals, the beneficial action of saliva following the licking of wounds. His belief was that saliva had some action on shed blood, probably by decreasing the coagulation time. The rapidity with which wounds in the buccal cavity stop bleeding, and the firm clot that occurs, seem to indicate that saliva has a coagulant action. Hunter, using his own blood and his own saliva, found a distinct reduction of the coagulation time. He also noticed that when gastric juice was added to the blood, coagulation at the end of fifty-five minutes was not complete, and that a jelly-like clot formed. By adding saliva to the tube containing the gastric juice and blood, the coagulation time could be brought down to twenty-six minutes, and in another experiment to seven minutes.

Hunter's conclusions, based on these experiments, are directed toward the treatment of hematemesis, and he believes that by augmenting the flow of saliva and using small doses of alkalies healing of the ulcer may be hastened.

Bellis, Birnbaum and Scott[3] repeated these experiments, using the blood of dogs obtained from the femoral artery, and noted a definite reduction of the coagulation time following the addition of saliva to the blood.

These experiments led us to seek some practical method by which use could be made of saliva in the surgery of the oral cavity and throat. As a matter of routine, we repeated those experiments, using human blood obtained from the median basilic vein, and saliva obtained from the blood donor as well as others.

Method and Experiments.—The coagulation time of the blood was obtained by the method of Howell.[4] In this manner the blood of healthy persons was found to cagulate in from ten to thirty minutes.

Saliva was obtained by chewing paraffin, and the blood was obtained as mentioned previously from the median basilic vein.

In the following experiments, we show the action of saliva as a coagulant and attempt to indicate some of the conditions necessary for its action, as well as to try in some way to determine the agent present in saliva that is responsible for its effect on blood coagulation.

EXPERIMENT No. 1.

SALIVA AS A COAGULANT.

Tube	Saliva	Blood	Coagulation Time	Remarks
1		2 cc.	15 minutes	Control
2	2 cc.	2 cc.	3 minutes	Spec. 1, saliva 24 hrs. old
3	2 cc.	2 cc.	3 minutes	Spec. 2, saliva 24 hrs. old
4	2 cc.	2 cc.	3 minutes	Spec. 3, mixed saliva of Spec. 1 and 2
5	2 cc.	2 cc.	3 minutes	Fresh saliva Spec. 1
6	2 cc.	2 cc.	3 minutes	Fresh saliva Spec. 2
7	2 cc.	2 cc.	3 minutes	Fresh saliva Spec. 3
8	2 cc.	2 cc.	3 minutes	Centrif. saliva for 10 minutes Spec. 1 sediment used

EXPERIMENT No. 1 (CONTINUED).

Tube	Saliva	Blood	Coagulation Time	Remarks
9	2 cc.	2 cc.	3 minutes	Centrif. saliva for 10 minutes Spec. 2 sediment used
10	2 cc.	2 cc.	3 minutes	Centrif. saliva for 10 minutes Spec. 3 sediment used
11	2 cc.	2 cc.	3 minutes	Spec. 1 centrif. saliva for 10 minutes Supernatant fluid
12	2 cc.	2 cc.	3 minutes	Same as 11—Spec. 2
13	2 cc.	2 cc.	3 minutes	Same as 11 and 12—Spec. 3

From experiment No. 1, it was definitely determined that coagulation time was greatly decreased by the addition of saliva. It was also noted that its action on coagulation was not materially different whether the saliva was fresh, exogenous, autogenous, centrifuged or twenty-four hours old, at room temperature or on ice. The method employed in this experiment is as follows:

All tubes were ready with the saliva placed in them, then blood was added immediately as it was taken from the vein. It was noted that, due perhaps to a difference in the specific gravity of the blood and saliva, the blood settled on the bottom of the tube. (The specific gravity of saliva as recorded is 1.002-1.008,[5] the specific gravity of blood plasma 1.0237, red blood cells 1.088.[6] However, this had no effect on coagulation time, because whatever constituent the saliva possesses that hastens coagulation, it certainly acts promptly, because the blood clotting begins just as soon as it comes in contact with the saliva. This may be seen in experiment No. 1.

A second series of tubes were prepared, using the same quantities of blood and saliva, and after the blood was added the tubes were gently shaken in order to mix the blood with the saliva, and it was found that coagulation occurred in about the same time.

EXPERIMENT NO. 2.

SALIVA AS A COAGULANT.

Tube	Saliva	Blood	Coagulation Time	Remarks
A.	2 cc. (24 hrs.)	+2 cc.	3 minutes	Specimen 1
B.	2 cc. (24 hrs.)	+2 cc.	3 minutes	Specimen 2
C.	2 cc. (24 hrs.)	+2 cc.	3 minutes	Mixed Spec. 1 and 2
D.	2 cc. (fresh saliva)	+2 cc.	3 minutes	Specimen 1
E.	2 cc. (fresh saliva)	+2 cc.	3 minutes	Specimen 2
F.		2 cc. whole blood control	10 minutes	(Control)

EXPERIMENT NO. 3.

SALIVA AS A COAGULANT.

Tube	Saliva	Blood	Coagulation Time	Remarks
1		2 cc.	10 minutes	Control
2	2 cc.	2 cc.	3 minutes	
3	2 cc.	2 cc.	3 minutes	Clot
4	2 cc.	2 cc.	3 minutes	became firmer
5	4 cc.	2 cc.	3 minutes	as
6	4 cc.	2 cc.	3 minutes	time increased
7	4 cc.	2 cc.	3 minutes	

In experiment No. 3, we repeated experiment No. 2, agitating each tube gently and the same decrease in the coagulation time was noted. The amount of saliva was also varied.

EXPERIMENT No. 4.

EFFECT OF GASTRIC ACIDITY ON SALIVA.

Tube	Saliva	Blood	Gastric Juice	Sodium Bicarb.	Normal Saline	Calcium Gluconate	Time, minutes	Remarks
1		2 cc.					10	Control
2	2 cc.	2 cc.					3	
3		2 cc.	2 No. 1				No clot after 20 min.	
4	2 cc.	2 cc.	2 No. 2				No clot after 20 min.	
5		2 cc.	2 No. 2				No clot after 20 min.	
6	2 cc.	2 cc.	2 No. 2				No clot after 20 min.	
7		2 cc.			2		10	
8	2 cc.	2 cc.			2		5	
9	4 cc.	2 cc.	2 No. 2				10 min. loose clot 20 min. loose clot	
10	6 cc.	2 cc.	2				3 min. clot began 10 min. loose clot 20 min. loose clot	
11	2 cc.	2 cc.	2	2 cc			3 min.	firm clot
12	2 cc.	2 cc.		2 cc			3 min.	firm clot
13	2 cc.	2 cc.				2 cc.	3 min.	firm clot

DISCUSSION OF EXPERIMENT NO. FOUR.

Gastric juice, No. 1—HCL 40.

Gastric juice, No. 2—HCL 30.

Tube No. 9 was divided into halves. To one half we added sodium bicarbonate and to the other calcium gluconate. Clotting was hastened and a very firm clot was formed immediately in both.

Sodium bicarbonate—3 1/1 in 30 cc. H_2O.

Calcium gluconate—gr. X in 30 cc. H_2O.

It was also noted that in tubes 9 and 10, when the amount of saliva was increased, the coagulation time decreased compared to tubes 4, 5 and 6, where coagulation had not started after twenty

minutes. In tube No. 9, when four centimeters of saliva was added to the gastric juice, a loose clot began to form in ten minutes. In tube No. 10, when six centimers of saliva was added, a loose clot began to form in three minutes. In tube 11, when sodium bicarbonate was added to neutralize the gastric acidity in the presence of saliva, a clot was formed in three minutes.

EXPERIMENT NO. 4A.

EFFECT OF GASTRIC ACIDITY.

Time	1	2	3	4	5	6
	2 cc. of saliva acidified + 2 cc. of blood	2 cc. of sodium bicarb. + 2 cc. of blood	2 cc. of calcium glucon. (sol. from powder) + 2 cc. of blood	2 cc. of calcium glucon. 10% sol. (Sandoz) + 2 cc. of blood	2 cc. of saliva whole + 2 cc. of blood control	Whole blood 2 cc. control
3 min.	no clot	no clot	no clot	no clot	clot	no clot
6 min.	no clot	no clot	firm clot	no clot	clot	no clot
8 min.	no clot	no clot	firm clot	no clot	clot	began to clot
11 min.	no clot	no clot	firm clot	no clot	clot	coagulated

In view of the finding of experiment No. 4, we repeated a portion of this experiment. (Experiment No. 4A).

Tube No. 1 acidified saliva (with free HCL), no clotting at the end of eleven minutes.

Tube No. 2, sodium bicarbonate and blood as a control on tube No. 11 and No. 12, experiment No. 4, no evidence of coagulation at the end of eleven minutes.

Tube No. 3, calcium gluconate (a saturated solution made from the powder), clotting took place at the end of six minutes. This was introduced as a check on tube No. 13, experiment No. 4. When the calcium is added in the presence of saliva, blood coagulation takes place in three minutes. When calcium gluconate is used alone coagulation takes place in six minutes.

In tube No. 2, two centimeters of a 10 per cent solution of calcium gluconate (Sandoz ampule form) was added to two centimeters of blood, and no evidence of coagulation occurred at the end of eleven minutes.

Experiment No. 5.

OPTIMUM PH AT WHICH SALIVA WILL ACT AS A COAGULANT.

In the following experiment, the Ph of the saliva was taken as soon as it was expectorated. The Ph was then varied by the addition of HCL until a series of tubes of the following reactions were otained: Ph 6.8, 6.6, 6.0, 5.4, 4.8. Acetic acid was added to saliva to obtain a Ph of 5.6 and to one tube lactic acid was added and a Ph of 5.2 was obtained.

Experiment: 2 cc. of saliva of the following reaction—2 cc. of blood:

Coag. time .	Normal saliva Ph 6.8	Norm. sal. after 3 hrs. Ph 6.6	With HCL Ph 6.0	With HCL Ph 5.4	With HCL Ph 4.8	Acetic Acid Ph 5.6	Lactic Acid Ph 5.2
2 min.	2 min.	2 minutes	2 min.	2 min.	5 min. very loose clot	5 min. fair clot	5 min. not firm clot
					10 min. loose clot	10 min. not firmly clotted	10 min. not firmly clotted
					15 min. very loose clot	fair clot	15 min. not firmly clotted

Control Blood 2 cc.—6 minutes (Howell).

Experiment No. 5A.

PH EXPERIMENT.

Time	1	2	3	4	5	6
	Ph 6.6	Ph 6.0	Ph 5.0	Ph 5.0	Control	Control
	saliva 2 cc. + blood 2 cc.	saliva 2 cc. + blood 2 cc.	saliva 2 cc. + blood 2 cc.	saliva 2 cc. + blood 2 cc.	saliva 2 cc. + blood 2 cc.	whole blood 2 cc.
3 min.	firm clot	no clot	no clot	no clot	clot	
5 min.	firm clot	firm clot	no clot	no clot	clot	
10 min.	firm clot	firm clot	no clot	no clot	clot	clot

Conclusion. Reaction of saliva:

As soon as the saliva was expectorated the Ph was taken and found to be Ph 6.8-6.6. This agrees with the work of Stan, quoted by Barlow,[7] who in a series of 610 cases found an average Ph of 6.6. There was no change in rapidity with which clotting was accelerated in range Ph 6.8-5.4. The reaction of the saliva was changed by the addition of HCL diluted. The coagulation within this range occurred in two minutes. (This patient had a rapid coagulation time—six minutes.) When the saliva was definitely acid, there was no clotting. Acetic acid Ph 5.6—fair clot formed at the end of fifteen minutes. Lactic acid produced a hemolysis of the blood and this did not aid clotting. Even when additional saliva was added there was no attempt at clot formation.

EXPERIMENT No. 6.

EFFFCT ON COAGULATION OF SALIVA OBTAINED AFTER VARIOUS PERIODS OF STIMULATION.

TWO CC. OF SALIVA—TWO CC. OF BLOOD AFTER PARAFFIN STIMULATION.

	Start saliva	15 min.	25 min.	35 min.	45 min.	55 min.	1 hour 20 min.	3 hrs.	4 hrs.
Time	3 min.	3 min.	3 min.	3 min.	3 min.	3 min.	3 min.	3 min.	3 min.

Control—6 minutes.

In this experiment, saliva was obtained by paraffin stimulation; all specimens were collected at intervals—at beginning of stimulation, 15 minutes, 25 minutes, 35 minutes, 45 minutes, 55 minutes, 1 hour and 20 minutes. It was found that clotting was hastened uniformly.

Paraffin was chewed for four hours, and all saliva obtained was expectorated and tested. At the end of three hours a specimen was taken and Ph of same was 6.6. Also, at the end of four hours a specimen was taken. It was noted that the saliva was much less viscid and more fluid, but when blood was added coagulation was hastened and took place in the same time interval.

EXPERIMENT No. 7.

STUDY OF SALIVA FILTRATES.

In the following experiment saliva was subjected to the process of filtration to see whether this would remove some agent from the saliva that was responsible for its coagulant action:

Time	1	2	3	4	5	6
	Saliva filtrate 2 cc. + blood 2 cc.	Saliva filtrate 2 cc. + blood 2 cc.	Saliva filtrate 2 cc. + blood 2 cc.	Whole saliva 2 cc. + blood 2 cc.	Water 2 cc. + blood 2 cc.	Whole blood control
2 min.	Blood coagulat. firm clot	Blood coagulat. firm clot	Blood coagulat. firm clot	Blood coagulat. firm clot	No Clot	
	Firm Clot	Firm Clot	Firm Clot	Firm Clot	No Clot	
	Firm Clot	Firm Clot	Firm Clot	Firm Clot	No Clot	
11 min.	Firm Clot	Firm Clot	Firm Clot	Firm Clot	Coagulat. jelly like same time on control	Firm clot

Discussion: Saliva filtrate acts similarly to whole saliva in hastening the coagulation time of the blood, illustrating that the process of filtration removes no beneficial agent from the saliva. This is in agreement with the work of Hunter,[2] who found that passage through a Berkefeld filter made no difference in the ability of saliva to hasten blood coagulation.

EXPERIMENT No. 8.

Coagul. time	OXIDIZING				REDUCING AGENTS		
	1	2	3	4	5	6	7
	Saliva 2 cc. + Na$_2$O$_3$ ½ cc. + blood 2 cc.	Saliva 2 cc. + Na$_2$O$_3$ 1 cc. + blood 2 cc.	Saliva 2 cc. + Na$_2$O$_3$ 2 cc. + blood 2 cc.	Saliva ½ cc. + Na$_2$O$_3$ 1 cc. + blood 2 cc.	Saliva 2 cc. + Na$_2$S$_2$04 2 cc. + blood 2 cc.	Saliva 2 cc. + Na$_2$S$_2$04 1 cc. + blood 2 cc.	Saliva 2 cc. + Na$_2$S$_2$04 ½ cc. + blood 2 cc.
2 min.	Bubbling small jelly like clot	Bubbling small jelly like clot	Gas bubbles jelly like clot	Gas bubbles jelly like clot	Loose clot	Loose clot	Loose clot
4 min.	Jelly like clot	Jelly like clot	Jelly like clot	Jelly like clot	Fairly firm clot	Fairly firm clot	Fairly firm clot
10 min.	Jelly like clot	Jelly like clot	Jelly like clot	Jelly like clot	Firm clot	Firm clot	Firm clot
15 min.	Loose clot	Loose clot	Loose clot	Loose clot	Firm clot	Firm clot	Firm clot

Control—12 minutes.

Discussion: Effect of oxidizing and reducing agents on saliva: Strong oxidizing agents do not seem to aid clotting, but a loose clot is formed. This clot is probably the result of a natural tendency or the inherent power of the blood to clot. The oxidizing agent seems to act as a slight hindrance to blood coagulation—only a loose clot formed at the end of fifteen minutes. The reducing substance seems to be of some aid to coagulation, as a firm clot had formed at the end of four minutes.

EXPERIMENT No. 9.

Attempt to fractionate saliva in order to determine the constituent responsible for the coagulating power it possesses:

In an attempt to determine the constituent of saliva that is responsible for its coagulant action on blood, we have tried to make up solutions of its various constituents in the proper percentage solution and test each one against blood to see if it possessed a coagulant action.

The composition of saliva was taken from Matthews,[5] as follows:

1. Water ....99.41 per cent
2. Solids .... 0.59 per cent
3. Mucin and epithelium........ 0.213 per cent
4. Soluble organic matter...... 0.142 per cent
5. Inorganic salts .... 0.129 per cent
6. KCNS .... 0.010-.00 per cent

In 1,000 parts of mineral ash there will be found the following constituents:

1. K...457.2
2. Na.. 95.9
3. CaO acid traces of Fe_2O_3............................ 50.11
4. MgO... 1.55
5. So_3... 63.8
6. P_2O_5 ....188.48
7. Cl...183.52

This experiment has been carried out in conjunction with Dr. William M. Malisoff, who used the following constituents as blanks to test their coagulant action against blood:

1. Water, which is 99.4 per cent of total composition of saliva when tested against blood (Experiment No. 7) has no coagulant action—in fact, it will produce hemolysis.

2. Experiment No. 9, which follows—three elements were used:

(a) Thiocyanate as sodium thiocyanate, .01 per cent solution (10 mgm. of sodium thiocyanate in 100 cc. of water—0.01 per cent).

(b) Calcium as calcium acetate, 35 mgm. of Ca. acetate in 100 cc. of water—(0.008 per cent Ca).

(c) Potassium as $KHCO_3$—250 mgm. $KHCO_3$ in 100 centimeters of water—(0.1 per cent of K).

The results noted are as follows:

Time	2 cc. of 0.01% Sodium Thiocyanate 2 cc. of blood	2 cc. of 0.008% of Ca (Calcium Acetate) 2 cc. of blood	2 cc. of $KHCO_3$ 0.1% of (K) 2 cc. of blood	2 cc. of whole saliva 1 blood 2 cc.	2 cc. of whole saliva 2 blood 2 cc.	2 cc. of autogenous saliva 3 blood 2 cc.
3 min.	No clot	No clot	No clot	Firm clot	Firm clot	Firm clot
5 min.	No clot	Slight evidence of clotting, jelly like	Slight evidence of clotting jelly like	Firm	Firm	Firm
7 min.	No clot	Not firm jelly like	Not firm jelly like	Firm	Firm	Firm
10 min.	No clot	Jelly like	Jelly like	Firm	Firm	Firm
12 min.	Fairly firm clot	Jelly like clot	Fairly firm clot	Firm	Firm	Firm

Control—Coagulation in 12 minutes.

Discussion: Thiocyanate blank No. 1 revealed no evidence of clotting until twelve minutes. Tube 2, the calcium blank—coagulation began at the end of five minutes, but a jelly-like clot was formed. Tube 3, the potassium blank—coagulation began at the end of five minutes, but a jelly-like clot was formed. It was noted

that in tubes 1, 2 and 3 clotting was complete at the end of twelve minutes, which was the coagulation time of this patient's blood. The solutions were probably of no aid to coagulation. In tubes 2 and 3 it was only of slight aid, but it is definite that they were of no hindrance to coagulation and that coagulation occurred in the same time as that of the control.

Tubes 4, 5 and 7 were controls, and coagulation took place in three minutes, showing again that saliva is of definite aid in the coagulation of the blood.

In tube No. 2—the calcium blank—which contained 0.008 per cent Ca in 100 cc. of water, which is the calcium content of saliva—it was noted that a jelly-like clot formed at the end of five minutes and that a firm like clot formed at the end of twelve minutes, which was the coagulation time of the control. The small percentage of calcium present in the saliva probably was not a factor in hastening the coagulation of the blood, as the control of whole saliva plus blood produced a firm clot in three minutes. The calcium content of blood plasma is noted as 9-11 mgm. per 100 cc. of blood, or approximately .01 per cent, which is a much greater percentage than that contained in the saliva. It was also shown by us in experiments Nos. 4 and 4A that large amounts of calcium, when added directly to whole blood, did not greatly hasten the coagulation time, unless it is added to the saliva and then mixed with the blood. It is probable then, although we are not prepared to make a definite statement, at this time, that the calcium content of saliva is not responsible for the coagulating power possessed by the saliva. Bellis et al.[3] consider it possible that the calcium is the agent that produces blood coagulation; our data seems to rule out this possibility.

EXPERIMENT No. 10A.

STUDY OF THE EFFECT OF THE POPULAR MOUTH WASHES AND GARGLES ON THE SALIVA, IN RELATION TO ITS EFFECT ON COAGULATION TIME.

	1	2	3	4	5	6
Coagulation time	Liquor Antisepticus Viridans 2 cc. + Saliva 2 cc. + Blood 2 cc.	Liquor Antisepticus Viridans 2 cc. + Blood 2 cc.	Liquor Antisepticus Alkaline, 2 cc. + Saliva 2 cc. + Blood 2 cc.	Liquor Antisepticus Alkaline, 2 cc. + Blood 2 cc.	Dobell's Solution 2 cc. + Saliva 2 cc. + Blood 2 cc.	Dobell's Solution 2 cc. + Blood 2 cc.
3 min.	Rather soft, loose blood clot	No clotting	No clotting	No clotting	Soft loose clot	No clot
5 min.	Fairly firm clot	No clot	No clot	No clot	Fairly firm clot	No clot
8 min.	Fairly firm clot	No clot	No clot	No clot	Fairly firm clot	No clot
11 min.	Fairly firm clot	No clot	Fairly firm, but this is time of control	No clot	Fairly firm clot	No clot

	7	8	9	10	11	12
Coagulation time	H₂O₂ 2 cc. Saliva 2 cc. + Blood 2 cc.	H₂O₂ Blood 2 cc.	Sodium Perborate 2 cc. + Saliva 2 cc. + Blood 2 cc.	Sodium Perborate 2 cc. + Blood 2 cc.	KMNO₄ 2 cc. Saliva 2 cc. + Blood 2 cc.	KMNO₄ 2 cc. Blood 2 cc.
3 min.	No clot	No clot	No clot	No clot	Blood seems to have settled on bottom of tube. No signs of clot	No clot
5 min.	No clot	No clot	No clot	No clot	No clot	No clot
8 min.	No clot	No clot	No clot	No clot	No clot	No clot
11 min.	No clot	No clot	No clot	No clot	Some slight evidence of coagulation at this time	

Blood control—Time of coagulation, 11 minutes.

Experiment No. 10B.

EFFECT OF MOUTH WASHES AND MOUTH ANTISEPTICS (continued).

Time	1	2	3	4	5	6
	Liquor Antisepti-cus + 2 cc. Saliva + 2 cc. Blood	Liquor Antisepti-cus + 2 cc. Blood	Liquor Antisepti-cus + 2 cc. Saliva + 2 cc. Blood	Liquor Antisepti-cus 2 cc. + 2 cc. Blood	Dobell's Solution 2 cc. + 2 cc. Saliva + 2 cc. Blood	Dobell's Solution 2 cc. + 2 cc. Blood
3 min.	No clot	No clot	No clot	No clot	No clot	No clot
5 min.	Soft clot	No clot	No clot	No clot	No clot	No clot
10 min.	Fairly firm clot	Semi-soft clot	Clot	No clot	Very soft clot	No clot

Control I.—Whole saliva, 2 cc. Whole blood, 2 cc. Blood coagulated in 3 minutes.

Time	7	8	9	10	11	12
	Lavoris 2 cc. + Saliva 2 cc. + Blood 2 cc.	Lavoris 2 cc. + Blood 2 cc.	Pepsodent 2 cc. + Saliva 2 cc. + Blood 2 cc.	Pepsodent + Blood 2 cc.	Vicks 2 cc. + Blood 2 cc. + Saliva 2 cc.	Vicks 2 cc. + Blood 2 cc.
3 min.	No clot	No clot	Blood hemolized	No clot	No clot Blood percipi-tates out	No clot
5 min.	No clot Blood seems hemolized	No clot	No clot	No clot	No clot	No clot
10 min.	No clot	No clot	No clot	No clot	No clot	No clot

Control II.—Whole blood, 2 cc. Coagulation time, 12 minutes.

EXPERIMENT No. 10C.

Time	1	2	3	4	5	6
	Mifflin 2 cc. + 2 cc. Saliva + 2 cc. Blood	Mifflin 2 cc. Blood 2 cc. +	Listerine 2 cc. + Blood 2 cc. + Saliva 2 cc.	Listerine 2 cc. + Blood 2 cc.	Glyco-thymoline 2 cc. + Saliva 2 cc. + Blood 2 cc.	Glyco-thymo-line 2 cc. + Blood 2 cc.
5 min.	No clot	No clot	No clot	No clot	No clot	No clot
7 min.	No clot	No clot	Soft jelly like clot	No clot	No clot	No clot
10 min.	No clot	No clot	Soft clot	No clot	Fairly firm clot	No clot
14 min.	No clot	No clot	Soft clot	No clot	Fairly firm clot	No clot
15 min.	No clot	No clot	Soft clot	No clot	Firm clot	No clot

Control I.—Saliva, 2 cc. Blood, 2 cc.—3 minutes.

Time	7	8	9	10	1	12
	Aspirin 2 cc. + Saliva 2 cc. + Blood 2 cc.	Aspirin 2 cc. + Blood 2 cc.	$KMNO_4$ 2 cc. + Blood 2 cc. + Saliva 2 cc.	$KMNO_4$ 2 cc. + Blood 2 cc.	Sodium Perborate 2 cc. + Saliva 2 cc. + Blood 2 cc.	Sodium Perbo-rate 2 cc. + Blood 2 cc.
5 min.	No clot	No clot	No clot	No clot	No clot	No clot
7 min.	No clot	No clot	No clot	No clot	No clot	No clot
10 min.	No clot	No clot	No clot	No clot	No clot	No clot
14 min.	No clot	No clot	No clot	No clot	No clot	No clot
15 min.	No clot	No clot	No clot	No clot	No clot	No clot

Control II.—Blood, 2 cc.—15 minutes.

Discussion: Experiments No. 10A-B-C.

In this experiment we attempted to make a study of popular mouth washes and antiseptics in relation to the effect they have upon saliva, or whether they hinder or aid it in its ability to accelerate coagulation of the blood. The method employed was as follows: The antiseptic was used in half strength—which is the usual dilution. The tubes were set up in the following manner:

To tube No. 1 we added 2 cc. of diluted antiseptic solution, 2 cc. of saliva and 2 cc. of whole blood. To the second tube we added 2 cc. of the diluted antiseptic and 2 cc. of blood. In addition,

we studied each antiseptic in its relation to the reaction to litmus paper. This was important, since we know that saliva is alkalin to litmus paper and acts best in medium of about Ph 6.8-6.6, as seen in experiment No. 5A. (Study of Ph). All the control tubes that were used, where the antiseptic solution was added to blood alone, showed no evidence of coagulation, indicating that these solutions in themselves are not coagulants. Where these solutions were mixed with saliva the results were as follows:

1. Liquor Antisepticus Viridans.—This is slightly acid to litmus paper, but in several trials we found that the blood coagulation occurred in five minutes—a soft clot—and by ten to eleven minutes (depending upon time of blood control), a firm clot resulted. This solution may not aid the saliva, but at least it does not hinder it. Its reaction is still within the range that will permit the saliva to hasten coagulation of the blood.

2. Liquor antisepticus alkalinus is definitely alkalin to litmus paper; we found, however, in several experiments, that clotting did not occur more rapidly than with the control. This solution, which theoretically should be of assistance to the saliva in producing blood coagulation, is in vitro of no apparent value.

3. Dobell's solution is definitely alkalin to litmus, but blood coagulation occurred after from three to five minutes, as shown in experiment No. 10A, and after ten minutes, as shown in experiment No. 10B. This is of some aid to blood coagulation and has no detrimental effect on blood coagulation.

4. Lavoris is acid in reaction and is of no aid to the saliva in producing blood coagulation. In fact, it seems to produce hemolysis and breaks up any slight clot that does form.

5. Pepsodent antiseptic is of no aid in blood coagulation and it also seems to break up the blood clot.

6. Vick's antiseptic is acid in reaction and is of no aid to coagulation and seems to break up the blood clot.

7. Mifflin is acid in reaction—no aid to coagulation and seems to break up the blood clot.

8. Listerine is acid in reaction and does not seem to disturb blood coagulation—a soft clot formed in seven minutes.

9. Glycothymoline is alkalin in reaction and seems to aid clot-

ting when mixed with the saliva. Blood coagulation occurred in seven minutes and clot seemed to get firmer.

10. Aspirin mouth wash (four tablets to a glass of water): There was no clotting in fifteen minutes. It is of no aid to blood clotting when added to the saliva.

11. Potassium permanganate is of no aid to blood coagulation and seems to produce hemolysis and breaks up the blood clot.

12. Sodium perborate is of no aid to blood coagulation. It seems to act similarly to hydrogen peroxide and breaks up any clot that has been formed.

Hydrogen peroxide seems to act similarly to sodium perborate in liberating the oxygen, thus breaking up the blood clot.

Discussion: It was seen that of the thirteen popular solutions often prescribed as a mouth wash following tonsillectomy, only the following were of any value in aiding the saliva in blood coagulation: (1) Liquor antisepticus viridans, (2) Dobell's solution, (3) Listerine, (4) Glycothymoline.

We have shown definitely that saliva is of definite aid in producing blood coagulation and wound healing. It seems to us that in prescribing a mouth wash for postoperative use one should recommend only those solutions that seem to aid coagulation. Of the four above mentioned, two were alkalin to litmus and the other two were slightly acid, no doubt within the range of Ph, as shown by us in a previous experiment. The solutions that are highly acid in reaction are of definite hindrance to blood coagulation and we believe should not be used.

EXPERIMENT No. 10E.

Use of blu-iodin (an aqueous colloidal iodin solution) and its effect on saliva as a blood coagulant:

Time	Colloidal Iodin 1:500 undiluted 2 cc. + blood	Colloidal Iodin 1:1000 2 cc. + 2 cc. blood	Colloidal Iodin 1:100 2 cc. + 2 cc. blood	Saliva 2 cc. + 2 cc. blood	Whole blood 2 cc.
2 min.	No clot	No clot	No clot	Clot	No clot
5 min.	No clot	No clot	No clot	Clot	No clot
8 min.	Jelly clot	Jelly clot	Jelly clot	Clot	No clot
10 min.	Jelly clot	Jelly clot	Jelly clot	Clot	Clot

Comment: Blu-iodin, an aqueous colloidal solution of iodin, does not hasten the coagulation time of blood as does saliva, but it is noted that clotting occurs in about the same time as the whole blood control; therefore, it really does not hinder the blood coagulation time.

EXPERIMENT No. 10F.

STUDY OF EFFECT OF CHEWING ASPERGUM ON BLOOD COAGULATION.

SERIES A.

Time	Saliva (2 cc.) Blood (2 cc.)	Aspergum (2 cc.) (First Spec.) Blood	Whole Blood 2 cc. Controll
2 min.	Blood clotted	No clot	No clot
5 min.	Clotted	No clot	No clot
8 min.	Clotted	No clot	No clot
10 min.	Clotted	No clot	Clotted

SERIES B.

Time	Saliva (2 cc.) Blood (2 cc.)	Aspergum (2 cc.) (First Spec.) Blood (2 cc.)	Whole Blood Controll 2 cc.
2 min.	Clotted	No clot	No clot
5 min.	Clotted	No clot	No clot
8 min.	Clotted	No clot	No clot
10 min.	Clotted	No clot	Clotted

SERIES C.

Time	Saliva (2 cc.) Blood (2 cc.)	Aspergum (2 cc.) (First Spec.) Blood (2 cc.)	Whole Blood Controll 2 cc.
2 min.	Clotted	No clot	No clot
5 min.	Clotted	No clot	No clot
8 min.	Clotted	No clot	No clot
10 min.	Clotted	No clot	No clot

Comment (Experiment No. 10F): It has been reported that numerous cases of secondary bleeding, postoperative, have resulted from the chewing of aspergum. The above experiment was carried out as follows: Tube 1 of each series was a control of whole saliva and blood. Tube 2 contained 2 cc. of saliva obtained by chewing aspergum, and the first specimen was used; this contained the greatest quantity of aspergum. Tube 3 was the whole blood control. It was noted that clotting occurred within two minutes in the whole saliva control. Coagulation was complete in ten minutes (Howell method in the blood control). There was no clotting in the tube in which the aspergum was mixed with blood (at the end of ten minutes). This is in accordance with our findings in a previous experiment, No. 10C, in which a solution of aspirin also prevented clotting of the blood. It would therefore appear that the chewing of aspergum, or the use of aspirin mouth washes and gargles should be avoided following tonsillectomy, due to their possible hemolyzing effect. In this experiment, we used the first mouthful of saliva obtained after chewing the aspergum for about one minute. This contained the greatest amount of aspirin.

EXPERIMENT No. 10G.

Comment (Experiment 10G): In this experiment, as in experiment No. 10F, we again used aspergum, after chewing for one minute and expectorating; 2 cc. of this was mixed with 2 cc. of blood, and, as noted in experiment No. 10F, no clotting occurred at the end of ten minutes. The saliva obtained after chewing aspergum for three minutes and five minutes, respectively, was collected; 2 cc. of this was mixed with 2 cc. of blood, and it was noted that a soft jelly clot was obtained at the end of five minutes. This, no doubt, was due to the fact that most of the aspirin contained in the aspergum was expectorated after the first one or two minutes of chewing. The whole saliva (2 cc.) and blood as a control produced clotting in two minutes, and the coagulation time of the whole blood was ten minutes. It may also be stated that after most of the aspergum was expectorated the small quantity that remained was neutralized by the saliva and therefore clotting occurred.

EXPERIMENT NO. 10G.

Time	2 cc. Saliva Blood 2 cc. Control	2 cc. Asper-gum Saliva 2 cc. Blood	2 cc. Asper-gum First Spec. 2 cc. Blood	2 cc. Asper-gum First Spec. 2 cc. Blood	Asper-gum after 3 min. 2 cc. Blood	Asper-gum after 3 min. 2 cc. Blood	Asper-gum after 3 min. 2 cc. Blood	Asper-gum after 5 min. 2 cc. Blood	Asper-gum after 5 min. 2 cc. Blood	Asper-gum after 5 min. 2 cc. Blood	2 cc. Blood Control
2 min.	Clot	No clot	No clot	No clot	No clot	No clot	No clot	No clot	No clot	No clot	No clot
5 min.	Clot	No clot	No clot	No clot	Soft jelly clot	Soft jelly clot	Soft jelly clot	Soft jelly clot	Soft jelly clot	Soft jelly clot	No clot
8 min.	Clot	No clot	No clot	No clot	Soft jelly clot	Soft jelly clot	Soft jelly clot	Soft jelly clot	Soft jelly clot	Soft jelly clot	No clot
10 min.	Clot	No clot	No clot	No clot	Soft jelly clot	Soft jelly clot	Soft jelly clot	Soft jelly clot	Soft jelly clot	Soft jelly clot	Clotted

Blood Control—Clotted in 10 minutes.

The following experiments were carried out by Dr. John A. Kolmer to determine whether saliva contains specific agglutinin for various organisms and erythrocytes.[9]

EXPERIMENT No. 11.

TOTAL BACTERIA IN SALIVA.

Saliva was plated out and it showed approximately 85,700 bacteria per centimeter. Paraffin was chewed for ten minutes and at the end of this time the saliva showed approximately 134,900 bacteria per centimeter. Ten minutes later a third specimen showed 64,900 bacteria, and ten minutes later a fourth specimen revealed 21,000 bacteria per centimeter.

EXPERIMENT No. 12.

AGGLUTINATION BY SALIVA.

Each of the four specimens showed the presence of large numbers of a nonhemolytic streptococcus, commonly designated as streptococcus salivarius.

Each of the four specimens of saliva were thoroughly centrifuged and the supernatant fluids were used in macroscopic agglutination tests against this streptococcus.

The final dilutions varied from 1:4 to 1:128, but none of the specimens of saliva produced any agglutination.

EXPERIMENT No. 13.

AGGLUTINATION OF ERYTHROCYTES BY SALIVA.

We then set up microscopic and macroscopic agglutination tests with each of the four saliva with a 1 per cent suspension of human erythrocytes.

The final dilutions were from 1:4 to 1:128, but all of the tests proved negative.

SUMMARY.

Discussion of experiments Nos. 11, 12 and 13:

1. Saliva collected with the aid of chewing showed a progressive diminution in the number of bacteria over the period of thirty minutes.

2. Saliva does not contain an agglutinin for streptococcus salivarius.

3. Saliva apparently does not contain an agglutinin for human erythrocytes. In this connection, however, it is to be kept in mind that the corpuscles of a single individual were employed, as it may be that saliva will contain agglutinin for corpuscles of one person and not from another.

These experiments were conducted with the saliva and corpuscles of the same individual.

4. It might well be that saliva hastens the coagulation of blood, but it is likely that it does not do so by favoring the agglutination of erythrocytes, according to the results of these experiments.

The following are a few of the case reports, showing the practical application of the experiments detailed above:

Experiment No. 14.

Patient—elderly male, was admitted to the ward of the Mt. Sinai Hospital, November 21, 1932, with a diagnosis of coronary thrombosis. Local tonsillectomy was performed, using novocain without adrenalin. Patient had a slight ooze from both fossae. A sponge was dipped into mixed saliva collected from the medical personnel of the clinic and inserted into both fossae. Oozing stopped immediately.

Experiment No. 15.

Patient—a youth, age 20, was admitted to the ward of the Mt. Sinai Hospital, November 21, 1932. Examination revealed large cryptic and infected tonsils. A tonsillectomy was performed under local anesthesia, using novocain, 1 per cent (no adrenalin). Free bleeding was present. A sponge soaked with saliva obtained from another group was inserted into both fossae. Bleeding was apparently controlled for the time, but an hour later free bleeding was noted. Considerable difficulty was encountered in controlling the hemorrhage. Finally, both fossae were painted with 50 per cent silver nitrate, supplemented with a hypodermic of morphin sulphate, grain, 1/6, and atropin sulphate, grain, 1/150, and bleeding was controlled.

Note: The two cases cited were given atropin before operation by the resident physician.

We concluded from the above experiments, the following:

1. Saliva is capable of controlling an ooze, but when there is arterial bleeding, it is necessary to clamp, tie, or suture all bleeding points or administer strong astringents.

Observation: It has been observed that when local tonsil cases are given atropin in doses that are sufficient to dry up the salivary secretions, the blood that is expectorated is free of saliva. It was noted that the blood remains fluid for several minutes after it is expectorated into the basin, clotting only after the expiration of the coagulation time of blood. When atropin is omitted, there is a free flow of saliva, and the blood coagulates almost as soon as it leaves the fossa and is mixed with the saliva. When the patient expectorated, we noted stringy coagulated masses of blood, bead-like in appearance.

Experiment No. 16.

In a group of ward patients admitted for tonsillectomy, we purposely omitted the preliminary preoperative procedure of administering a hypodermic of morphin and atropin sulphate. There was considerable salivation following the intra-oral manipulations, and what bleeding did occur, seemed to subside. When the patients expectorated, the blood was beady in appearance, partially clotted and mixed with the saliva.

Experiment No. 17.

Another group of adults was admitted to the hospital for tonsillectomy. These patients were given atropin sulphate, grains 1/75, hypodermically, one-half hour before the operation. We noted that their mouths were very dry prior to operation, and no salivation occurred during operation. In all of these cases, the blood expectorated was in a fluid state. In some the clotting was almost spontaneous when it reached the basin. We purposely selected this group of patients for this particular experiment because they all had a rapid coagulation time. We failed to note the bead-like blood clots intermixed with the saliva. The clotting here was due to the inherent coagulating properties of shed blood rather than to the saliva.

Experiment No. 18.

A local tonsillectomy was performed on a patient at 10 A. M. At 4 P. M. examination revealed both fossæ were dry. At 7 P. M. we noted a large laminated clot in the right fossa. This patient was salivating freely and when the clot was removed, a bleeding point was noted. It may be stated that although the salivary secretions are not entirely adequate in controlling bleeding, still the action of the saliva in mixing with the blood to form a laminated clot, prevents excessive bleeding.

Experiment No. 19.

Eight tonsillectomies and adenoidectomies were performed under general anesthesia. The patients were given atropin sulphate preoperatively. In all of these cases, the mouths were dry and free of saliva. It was noted that when oozing did occur, there was very little attempt at clotting. This we attributed to the fact that saliva, which is an aid to coagulation, was absent.

Coagulation in and about the oral cavity is dependent upon the following factors:

(a) The inherent coagulating properties of the blood.

(b) It seems that with constant and active salivation, saliva forms a film over the bleeding area and each film acts in the coagulation process thus producing a laminated clot.

Experiment No. 20.

A tonsillectomy and adenoidectomy was performed under general anesthesia. Some venous ooze was present. By means of a powder blower, sodium bicarbonate was introduced into the left tonsillar fossa. It did not seem to have any effect as a coagulant. However, coagulation ensued when it mixed with the saliva.

It might be stated that in cases where vomiting occurs and bleeding is persistent, especially where the vomitus is definitely acid, it might be advisable to introduce sodium bicarbonate into the fossae so as to neutralize

the acid vomitus. Thereby coagulation will be aided. (Litmus to test reaction of fossae.)

According to our experimental data, $NaHCO_3$ given by mouth, pre-operatively, neutralizes the gastric acidity and indirectly aids coagulation.

If vomitus acts as a coagulant, it is probably due to the following:

1. Absence of free HCL in vomitus (gastric juice).

2. The excess saliva as it is swallowed neutralizes the acidity in the stomach, and the so-called effect of the vomitus upon coagulation is really due to the saliva.

3. Where vomitus occurs during the induction of the anesthetic, the greater portion of the gastric contents are emitted. The remaining acid is probably neutralized by the saliva as it is swallowed during the induction of the anesthesia and during the operation. The beneficial effect of the gastric juice on coagulation is probably due to the alkalinization of the gastric contents by the saliva.

4. Reflex flow of saliva occurs at beginning of vomiting.[10]

EXPERIMENT No. 21.

A tonsillectomy with a Tydings snare was performed under local anesthesia. As the tonsils were dissected from the fossae from above downward, repeated insufflations of calcium gluconate powder are made into the fossae until the tonsils were brought to the inferior portion of the fossae; insufflation was repeated several times and the raw surface of the fossae were completely covered with a film of calcium gluconate powder. The fossae were completely dry. In another instance where there was free oozing in the upper part of the fossa, a fresh insufflation of calcium gluconate seemed to control the bleeding.

Note: According to experiment No. 4—test tube No. 13—where calcium gluconate was added to the blood and saliva, we found that coagulation had taken place within three minutes on a ten-minute control.

In vivo, calcium gluconate mixed with blood and saliva seems to shorten the bleeding time, as by the time the tonsils were amputated, the fossae were completely dry.

EXPERIMENT No. 22.

We noted that moistening cotton balls in saline solution and dipping them into calcium gluconate powder, and applying them directly to the fossa, is a gentler and more efficient method than the powder blower. By this method, several applications were made and when the tonsil was gradually dissected and enucleated the fossa was dry.

In some instances, where oozing continues, another application of calcium gluconate powder may control it. Incidentally, it might be well to mention that calcium gluconate seems to abate post-operative edema of the fossa. We should also like to add that the moist method is safer than employing the powder insufflations. It lessens the danger of the powder being inhaled into the bronchi.

After tonsil enucleation, suction should be limited to the pharynx and not extended to the operative field in order to prevent the removal of the protective film of saliva in the tonsillar fossæ. When suction is employed, it is best to use it intermittently rather than continuously. In that way we allow the saliva sufficient time to bathe the fossæ. Should there be bleeding, this protective layer of saliva will aid coagulation.

Experiment No. 23.

PREOPERATIVE PREPARATION OF PATIENTS.

As has been stated at the beginning of this paper, vomitus is not an aid to clotting, as is popularly supposed, except that, perhaps, for the reflex flow of saliva that occurs at the onset of vomiting.[10]

We studied a series of cases with Dr. Samuel Immerman, and we attempted to remove all the fasting gastric contents so as to have an idea as to the quantity and acidity that was present. Also to note the effect of a preoperative dose of atropin on free and total acidity of the contents.

The results are as follows:

Experiment No. 23.

Case	Fasting Free	Before Total	Atropine Amount	Fasting Free	After Total	Atropine Amount
1	67	95	13 cc.	64	112	50 cc.
1				50	86	20 cc.
2	0	97	174 cc.			
3	51	76	100 cc.	12	54	5 cc.
3	12	47	7 cc.	8	46	6 cc.
4	8	45		7	53	8 cc.
5	54	106	40 cc.	30	60	18 cc.
6	5	40	10 cc.	0	65	2 cc.
7	15	41	174 cc.	31	55	48 cc.

In this series a total of fifteen fasting extractions were observed.

1. An average fasting content of 38 cubic centimeters.
2. Every patient showed a definite acidity, free and total.

3. Each patient was given 1/75 of a grain of atropin hypodermically, and the fasting extraction was made one-half hour later. We are sure that the dose administered was of sufficient strength, because most of the patients manifested the physiologic effects of atropin, as evidenced by rapid pulse, dry mouth, dilated pupils and flushing of face. However, it did not have an appreciable effect on the gastric juice.

With the above findings in view, and since we have shown that the acidity of the gastric juice and the vomitus is sufficient to delay the coagulation time from twenty to fifty-six minutes. when in direct contact, it was therefore concluded that when the vomitus bathes the bleeding surfaces of the fossæ with the gastric contents, this factor alone is sufficient to promote tonsillar bleeding. We have also shown that by neutralizing the acidity with saliva or sodium bicarbonate, we are able to bring the coagulation time down to the normal three to five minute limit.

With this observation in mind, we would suggest that the following preoperative routine be carried out:

1. One hour prior to operation, each patient is given a dose of sodium bicarbonate (gr. V to X to children and up to $\mathfrak{Z}$ ÷ in adults), with a view towards neutralizing the gastric acidity.

2. Calcium gluconate, grains X, by mouth.

3. Each patient is given a piece of paraffin to chew and is instructed to swallow all the saliva that was stimulated by his chewing.

With the above therapeutic measures, we accomplish the following:

(a) We neutralize gastric acidity.

(b) There is sufficient alkalin residue to neutralize further acidity.

(c) Direct bathing of the mouth and tonsillar fossæ with the saliva.

In fact, we encourage vomiting while the patient is still on the operating table. This is accomplished by permitting the patient to come out of the anesthetic and deliberately gagging him. All told, these factors have an efficacious effect in aiding coagulation.

That saliva does have an alkalinizing power, we have illustrated in experiment, No. 24, which follows:

Experiment No. 24.

Saliva was placed in a burette and used to titrate against 1 cc. of gastric juice, using Topfer's reagent as an indicator.

Case T. C., No. 1.

Free acid 24, using saliva.

Note—We used 7 cc. less of saliva than 1/10 N. NAOH to neutralize the acidity.

This illustrates the degree of alkalinity that is possessed by the saliva and its aid in neutralizing gastric acidity.

We also know that a normal individual will secrete about 25 cc. or more of saliva in fifteen minutes by artificial stimulation. If this were swallowed, as suggested above, or collected preoperatively in a proper container, one could utilize this material. Since saliva has a definite healing and positive coagulating power, in order to encourage coagulation where bleeding is noted, successful results can be attained by dipping sponges in the saliva and inserting them into the fossæ, or one can instill the saliva either with a pipette or medicine dropper directly into the operative field.

After tonsillectomy we have noted that a secondary hemorrhage often occurs on the fifth or seventh day. This is invariably due to a slough. In these cases there is generally a history of excessive salivation during the first few postoperative days, and this is followed by a complete cessation of the salivary secretions. The patients invariably complain of pain over the parotids, submaxillary regions, and often tenderness on palpation of these glands, due to a congestion and overactivity. The discomfort may be relieved by administering small doses of atropin during the first few postoperative days and in turn the glands will be rested, as it were. This precaution would insure a constant flow of saliva. We believe that the salivary flow is beneficial in preventing excessive dryness, slough and secondary hemorrhage, as the slough, no doubt, is the result of dryness. It is, therefore, needless to reiterate the important rôle that saliva plays when it comes in contact with the blood.

For many years we have had the privilege of observing and correlating these observations in Dr. William Ersner's practice.

His belief and experience taught him that all tooth sockets should be filled with blood clot immediately after extraction, or otherwise dry sockets may result. By adhering to this technic, one may be assured of primary union and rapid wound healing. This procedure invariably insures early dental prosthesis. It is advisable to abstain from using a mouth wash immediately after extraction, because it has a tendency to break up any blood clot formation, and in turn it prolongs postoperative bleeding. It is best, primarily, to utilize the natural flow of saliva, because it possesses certain inherent qualities that are of aid to coagulation. Saliva is slightly germicidal, it acts as a cleanser, promotes healing and is a most effective coagulant when in contact with the blood. A mouth wash, when followed immediately after oral surgery, has a tendency to destroy these attributes. We noted that when the socket is allowed to be bathed by a free flow of saliva a blood clot forms more rapidly. It is also observed that by avoiding suction around the operative area a clot formed sooner and the wound healed more readily. These observations we know occur in vivo, but we could not explain them adequately until this research problem was completed. We now know the part saliva plays in coagulation. As stated previously, we are aware of the detrimental results caused by the anticoagulant mouth washes (experiment No. 10). The various experiments have led us to believe that healing in the oral cavity is best accomplished by the existing qualities of the saliva.

To diverge somewhat from the subject, if we may, we should like to call your attention to the blood clot dressing in mastoid surgery. Dr. George M. Coates and his co-workers, who have been exponents of this method, have found that primary union will often result when the blood clot is properly employed. Dr. Coates, in his teaching, lays special stress on this question and emphasizes that one should prevent all chemicals or solutions from entering the wound. As he states, many a good blood clot has been destroyed by an inexperienced assistant, because he used antiseptics indiscriminately either by direct application or by irrigation of the wound. Therefore, what holds true in one wound would naturally follow in another. However, there is this difference: In the mastoid the blood clots at its normal coagulation

time, while in the oral cavity the saliva decreases the bleeding time.

To reiterate, it is best to avoid the use of chemicals after oral surgery and depend upon the inherent qualities of the saliva to aid coagulation, as the chemicals have a tendency to break up the blood clot rather than aid it.

SUMMARY AND CONCLUSION.

The experiments given above have been detailed to show the effect of saliva on wound healing and in decreasing the coagulation time of the blood. Each experiment has been given in detail in the text with discussion of each. The summary of the experiments and their results follows:

1. Saliva when added to blood in vitro has produced in numerous trials a reduction of the blood coagulation time from ten to fifteen minutes to two to three minutes.

2. The saliva, whether autogenous, exogenous, mixed from various individuals, centrifuged, using supernatant fluid or the sediment, has the same effect in producing a reduction of the coagulation time. Since the earlier experiments were concluded, we found that saliva that has been kept on ice from twenty-four to forty-eight hours, or at room temperature for this length of time, had the same effect in decreasing the coagulation time, showing again that whatever agent is present does not become inert.

3. Fresh saliva is slightly acid to phenolphthalein and alkalin to litmus. The reaction of saliva has been determined by us as Ph 6.8 to 6.6, corrobating the results of other workers. We have noted that this is the optimum reaction at which saliva aids blood coagulation. However, in a series of experiments as shown, coagulation will take place in a range of Ph 6.8 to 4.8. Acidification of saliva below Ph 4.8 is sufficient to prevent the saliva in aiding coagulation.

4. Several subjects were given paraffin to chew for four-hour periods. This saliva was tested for its coagulating power at various time intervals. It was noted that saliva, after continued stimulation, is less viscid, but, nevertheless, retained its coagulating power and the Ph of the saliva did not vary.

5. Filtered saliva possesses the same coagulant action as whole saliva.

6. Utilizing the analysis of the composition of saliva, as quoted by Mathews, we prepared aqueous solutions of the following inorganic constituents, namely, thiocyanate, calcium and potassium in the same percentages as found in saliva. These were used individually and combined to test their coagulating action on blood. It was found that they did not possess coagulant action, and we therefore concluded that they are not the factors responsible for the coagulant power of saliva.

7. Bacteriologic experiments conducted show that saliva acts as a diluent of the organisms present in the mouth, and that as increased flow of saliva such as is present following intra-oral manipulations or after artificial stimulation seems to wash the organisms from the mucous membranes of the oral cavity.

From the knowledge gained from these experiments, as well as from the frequent clinical trials, we would suggest the following preoperative preparation for patients who are to undergo oral, dental or throat surgery, under general anesthesia, where vomiting is apt to occur. We have previously shown that the acid vomitus will delay the coagulation for a considerable length of time.

(a) Administration of sodium bicarbonate to neutralize gastric acidity.

(b) That the patient be given paraffin to chew, or some other inert substance that will stimulate the salivary secretions, and this saliva to be swallowed, so that its presence will alkalinize the gastric acidity as well as aid blood coagulation.

In closing, we desire at this time to mention a few men who have figured prominently in the various research problems. These problems were a contributory factor in making this work possible. We therefore wish to express our appreciation and gratitude to Drs. Malisoff, Gruskin, Immerman and Kolmer for their invaluable aid and co-operation, and to the student body who so liberally contributed their blood for this work.

1915 Spruce St.

BIBLIOGRAPHY.

1. Grevin: Saliva in the Treatment of Wounds. J. de med. de Bourdeaux, 89:140 (May, 1918).

2. Hunter, John B.: The Action of Saliva and Gastric Juice on the Clotting of Blood. Brit. Journal Surg., 16:203 (October, 1928).

3. Bellis, C. J.: Birnbaum, W.; Scott, F. H.: Effect of Saliva on Coagulation of Blood. Proc. Soc. Exp. Biol. and Med., 29:1107 (June, 1932).

4. Todd and Sanford: Textbook, "Clinical Diagnosis by Laboratory Methods."

5. Mathews, A. P.: Physiological Chemistry, p. 333.

6. Ibid, p. 477.

7. Barlow, R. A.: ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY. 1021 (Dec., 1926).

8. Mathews, A. P.: Physiol. Chemistry, p. 333. Composition of Saliva.

9. Incompleted Work, Collaborating with Dr. John A. Kolmer.

10. Howell: Textbook of Physiology, 11th Edition. W. B. Saunders, p. 75.

11. Incompleted Work: Ersner, Immerman, Myers. Effect of Preoperative Atropin Dosage on Amount and Composition of Gastric Juice.

XI.

THE INVOLUNTARY NERVOUS SYSTEM IN ITS RELATION TO OTOLARYNGOLOGY.

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The lowest forms of life exist without nervous control. Higher forms of life retain this intrinsic power of cell metabolism according to the electrolytic state and the ionic content of their cells. With the differentiation of cellular life there arose the need for a correlation between the various cells. First in the evolutionary process appeared the chemical correlating agents as exemplified in the internal glandular secretions and retained in the higher forms of life.

The complexities increased as the organism progressed ,and there came a demand for a correlation which would give a more rapid response to external environments and to the needs of internal metabolism. Later came the development of a system that can receive impulses from and transmit impulses to the cells in all parts of the body. In the lower orders of life this is accomplished through reflexes independent of any volitional capabilities of the organism.

As ascent is made in the scale of life, this correlating system is divided into two divisions, the voluntary and the involuntary nervous system.

The voluntary or somatic system has been developed particularly for the struggle for existence, for food necessities and for protection against external enemies. While functioning through simple reflexes it is primarily supervised by the conscious will, receiving afferent impulses and sending efferent impulses to the skeletal muscles.

The second division is known as the autonomic, vegetative, interofective, or involuntary system. No one term adequately describes its activities. While exercising control over reproduction, nutrition and growth, it is subject to mechanical, chemical and

psychic stimulation. It does play its part in protecting the individual when exposed to external dangers, functioning beyond the limits of ordinary cell metabolism. Autonomically it governs the activities of smooth muscles and glands, and functions to preserve homeostasis, and constancy of internal economy. This second division manifests itself in a definite vegetative manner, affecting blood pressure, vasodilation, digestion and the like, yet it does not constitute a complete system. The complete arc is dependent upon the afferent fibers of the somatic system, and upon their impulses depends much of the so-called tonicity of the involuntary system. Perhaps Cannon's term interofective, in contradistinction to the voluntary system, which he terms exterofective, gives a definition that more fully covers the system's functional activities.

The responses in the two systems materially differ. In the somatic system a single impulse is sufficient, but in the involuntary (interofective) there is often a delayed and slower response.

The involuntary (interofective) system controls and regulates vital functions, but not entirely independent of central nervous control. The responses in the somatic system are limited, while in the involuntary (interofective) they are more diffuse. It is this diffuse response to afferent impulses, explained anatomically, that facilitates the maintenance of homeostasis.

Viscera show a double innervation by the involuntary (interofective) system. This double innervation comes from its two subdivisions, the thoracolumbar or sympathetic, and the craniosacral or parasympathetic. They show essential anatomic differences and are generally opposed to each other in their efferent impulses.

The cells of all nerve tissue have a similar morphologic structure; that of neurone, axone and dendrites. The afferent fibers coming to the spinal column enter by the posterior root, with the nutrient cell being in the posterior ganglion. Every part of the body is supplied by some sort of sensory nerve. Sensory fibers are stimulated through receptors which may require actual contact as in touch, temperature and taste or be stimulated through space as with sight and hearing. Reflexes are maintained mainly through the spinal cord and lower parts of the brain. Afferent impulses in the somatic system may pass directly to cerebral cen-

While the sympathetic impulse is diffused, the parasympathetic activities operate at different levels in various viscera and parts. The various centers are widely separated. None are entirely without the influence of the central nervous system. Those in the spinal cord are much less subject to this influence than the parasympathetic neurones. In fact, the enteric plexuses have the capacity to carry out coordinated nervous reflexes independent of the central system.

The antagonistic activities of the two systems are seen in the striated heart muscle, where the vagus (parasympathetic) inhibits and the sympathetic accelerates. In the intestines the reverse occurs, the vagus accelerating and the sympathetic retarding.

ters without causing a definite reflex reaction. The axone of the somatic afferent fiber ends in synaptic connection with the connector neurone in the posterior horn of the cord; the short connector neurone in turn ends in synaptic connection with the efferent neurone in the anterior horn, from which its axone leaves by the anterior root of the spinal nerve to innervate the skeletal muscles.

The involuntary (interofective) system differs primarily from the voluntary system in that the connector neurone lies in the lateral horn of the cord and does not meet with another neurone in the spinal column but sends its axone out through the anterior division of the spinal nerve, where between the eighth cervical and second lumbar vertebræ the fibers make connection with ganglia arranged segmentally and resting alongside the vertebral column. The fibers may or may not end in the first ganglion entered; they may give off branches and go, either up or down, to other ganglia. Together with the ganglia they form the sympathetic chain. The fibers from the cord to the ganglia are always myelinated and spoken of as the white rami or preganglionic fibers. In contradistinction, the majority of fibers leaving the ganglia are gray fibers, unmyelinated and known as postganglionic fibers.

Afferent fibers entering the spinal column may pass either up or down in the cord, but it is the outlying neurone in the sympathetic chain that functions to multiply the channels of efferent impulses. The overlapping up and down through the ganglia by the preganglionic fibers is responsible for the diffuse effect of the nerve cell discharges.

Further diffusion is accomplished by the various plexuses as the pulmonary, cardiac, coeliac, hypogastric and pelvic. The cardiac, pulmonary and coeliac are intimately connected with the vagus nerve. The hypogastric plexus forms the connecting link between the coeliac and the sacral plexuses. Subordinate plexuses follow and surround the arteries as they leave the aorta. Enteric plexuses extend from the upper level of the esophagus to the anus. The cephalic sympathetic plexuses originate from the superior cervical ganglion.

In the cervical region the sympathetic chain has differences from that in the thoracolumbar region. The white communicating rami or preganglionic fibers are limited to the region from the first dorsal to the second or third lumbar, while gray communicating rami pass from all the ganglia to the spinal nerve roots. In the cervical region the ganglia instead of being arranged segmentally are three in number, superior, middle and inferior, the latter frequently fusing with the first dorsal ganglion. The trunk and three ganglia contain myelinated and unmyelinated fibers. There are no visceral afferent fibers beyond the middle cervical ganglion, and stimulation of the cervical sympathetic trunk by not eliciting any general body reflexes demonstrates an absence of afferent fibers. Langley showed that section of the cervical sympathetic centrally to the superior ganglion caused complete degeneration of the fibers up to the ganglion, but peripherally to the ganglion there was no evidence of degeneration. Ransom and Billingsly confirmed this absence of afferent fibers in the cervical sympathetic chain. The white communicating rami are the only efferent connection between the cerebrospinal system and the sympathetic ganglia. These are not present in the cervical sympathetic chain where only gray rami pass from the ganglia to the spinal nerve roots. The only preganglionic fibers in the cervical sympathetic chain all pass through the stellate ganglion, and the source of those going to the superior cervical ganglion may be as low as the seventh dorsal segment.

The inferior cervical ganglion has gray connecting rami to the seventh and eighth and at times to the sixth cervical and first dorsal nerve roots. It originates the inferior cardiac nerve, has a branch connecting with the recurrent laryngeal nerve and sends fibers to plexuses about the carotid, subclavian, vertebral and internal mammary arteries.

The middle cervical ganglion communicates in like manner with spinal nerve roots of the fifth and sixth, and occasionally with the fourth and seventh cervical nerves. It originates the middle cardiac nerve and has branches going to the thyroid and the subclavian arteries.

The preganglionic fibers from the first four or five thoracic segments include all the sympathetic innervation of the eye, face,

head, neck and heart, lungs and upper extremities. The cephalic sympathetic innervation all comes from the superior cervical ganglion. The only preganglionic fibers in the cervical chain pass through the stellate ganglion and originate in the dorsal segments of the cord. Those for the superior ganglion are usually the first, second and third dorsal, but may extend as low as the seventh. From the superior cervical ganglion there are gray rami to the first four cervical nerves. It originates the superior cardiac nerve and has a branch or branches to the twelfth, to the jugular and nodose ganglia of the tenth, to the petrosal ganglion of the ninth, to the esophagus, to the phrenic, to the superior laryngeal, rarely the recurrent laryngeal, and connects with the ascending pharyngeal, carotid and external maxillary plexuses. The branches to the internal carotid artery form the internal carotid plexus and constitute the sympathetic extensions into the head. The branches of the superior cervical ganglion about the internal carotid artery continue within the skull where there is a separation into the carotid and cavernous plexuses. The latter is concerned principally with the ocular tissues and the hypophysis. The tympanic plexus in the median wall of the middle ear is formed by sympathetic fibers through the caroticotympanic branch from the internal carotid plexus and a branch from the petrosal ganglion.

The cephalic ganglia are in relation to the third and fifth nerves but interofectively functionate through the nerves from which they receive their preganglionic fibers. The ciliary, sphenopalatine, submaxillary, otic, geniculate, petrosal, jugular and nodose ganglia receive sympathetic and parasympathetic fibers.

The bulb and lower brain are merely expansions of the spinal cord. In each are located reflex centers that control physiologic functions independent of voluntary effort. The vast and intricate arrangement of nerve tracts in the brain and spinal cord obscure analogies between the sympathetic and parasympathetic systems. Both arise from corresponding groups of cells in gray matter with preganglionic elements of medullated fibers and connector elements which do not run directly to muscles or glands but end in an effector element in a ganglion. In the sympathetic system the ganglion is located extraneurally but in immediate proximity to the cord. In the parasympathetic system the preganglionic

fibers are sent to their peripheral destination, the true cells of the parasympathetics being in or near the tissues of the organ innervated. The involuntary outflow from the sacral region resembles that from the craniobulbar in that the fibers do not pass through segmental ganglia but unite to form the pelvic nerve. This arrangement of the parasympathetics, craniobulbar and sacral permits of a more selective action of efferent impulses. Through them there may be variations in the impulses transmitted —that is, one viscus or part may be separately influenced through its parasympathetic innervation. This is in contrast to the diffused impulses received from the sympathetic division.

Cannon has aptly compared the action of the sympathetic fibers to the soft and loud pedals of the piano, and that of the parasympathetics to the individual keys.

Neurones in the medulla, diencephalon and walls of the third ventricle possess certain nerve activating powers in relation to vasoconstriction, trophic regulation of the skin and fatty tissues, the smooth musculature of the eye, the lacrimal and salivary glands, carbohydrate and protein metabolism, body temperature and perhaps blood sugar. Their status as definite control centers has not been proven.

While the pathways from the diencephalon to the medulla are not well established, the clinical and experimental evidence of such connections to visceral efferent nuclei cannot be denied. Experimentally vasomotor conductive pathways have been demonstrated in the cord without their exact course being known. There is no doubt as to the regulatory influences exerted by the medulla and the diencephalon, yet no definite system of descending fibers in the spinal cord are known.

The double innervation by the sympathetic and parasympathetic systems must have a purpose. It is conceded that most of the impulses from both are efferent in nature. It is assumed that what involuntary control of body balance exists is maintained by a balance of impulses through the sympathetic and parasympathetic fibers. Also by some that when there is need for increased sympathetic tonus there is a corresponding decrease in parasympathetic tonus, and vice versa.

While the sympathetic impulse is diffused, the parasympathetic activities operate at different levels in various viscera and parts. The various centers are widely separated. None are entirely without the influence of the central nervous system. Those in the spinal cord are much less subject to this influence than the parasympathetic neurones. In fact, the enteric plexuses have the capacity to carry out coordinated nervous reflexes independent of the central system.

The antagonistic activities of the two systems are seen in the striated heart muscle, where the vagus (parasympathetic) inhibits and the sympathetic accelerates. In the intestines the reverse occurs, the vagus accelerating and the sympathetic retarding. The sympathetics constrict the blood vessels and if one acknowledges parasympathetic innervation, it is certainly one of dilation. Otherwise the contractility is affected by variations in sympathetic impulses. The innervation of the sweat glands through the sympathetic system is well established, but there is no proof that they are supplied by the parasympathetics. The pupils contract through impulses from the midbrain by means of preganglionic fibers in the third nerve, but dilate through preganglionic fibers coming from the upper thoracic segments via the superior cervical ganglion.

The emotions or cortical processes affect the vasomotor mechanism and each in a different way, worry by vasoconstriction, and joy by vasodilation; likewise is the somatic system affected, pleasure bringing a buoyant posture and grief a drooping type.

The diffuse action of the sympathetic nerves acts to maintain a constant internal environment as seen in the contraction of peripheral vessels relating to cold and the acceleration of the heart and pulmonary activities reacting to excessive muscular work, thereby counteracting the reduction of blood sugar. While the sympathetic system is concerned with vital activities, Cannon has shown that it is not essential to life in a protected environment. Parturition and normal growth can take place without sympathetic innervation. Cannon has demonstrated these facts, but he has also shown that there is marked reduction in prolonged ability to work, that there is a lack of preparation for lactation and marked defects noted when exposed to high or low tempera-

tures. His experiments have shown that an animal can live more or less normally without sympathetic innervation in a protected environment but that he would be in a precarious state was he exposed to the elements, the struggle for food and the dangers from hemorrhage and external enemies. The liberation of sugar and the increase of red blood cells through splenic co-operation under the stimulation of excitement would be lacking.

The sympathetic system as a protector functions essentially in a reflex manner. It acts promptly to prevent serious changes in the body. It makes more readily available the potential energies of the tissues. In doing so it is catabolic in action and serves as a direct adjuvant to the somatic system in protecting the individual.

The cranial and sacral (parasympathetic) divisions are essentially conservative and upbuilding. There is here an ability to particularize activities which are to a greater extent influenced by cerebral control. In the sacral division the impulses act to empty hollow organs that are periodically filled, the rectum, colon and bladder, the afferent impulse being supplied by the stretching of the viscera by their contents. The uterus which some claim to have only sympathetic innervation can undergo parturition without mid-dorsal influence.

Gaskell has stated that afferent fibers have their nutrient center in the posterior root ganglia and that the course of sensory fibers for all sensory nerves is the same and that all are medullated. From this he concludes that all nonmedullated fibers are efferent and that the sympathetic system does not constitute a complex system but consists only of excitor fibers. There is not universal acceptance of this dictum, nor is there proof that ganglia of the involuntary (interofective) system include afferent neurones, thereby constituting a reflex system.

Quoting J. T. McClintock, professor of physiology at the University of Iowa: "Whether or not the autonomic system has afferent fibers or is purely efferent is a matter of definition. It can be and is by some defined as a purely efferent system; then the afferent fibers which may accompany the nerve would be classed as somatic. For example, the mixed character of the vagus with afferent fibers from the viscera. There are afferent

fibers in the sphlanchnics, and whether or not you call them sympathetic or somatic depends upon your definitions, as histologically they cannot be differentiated."

Swift and Flothow (Medical Clinics of N. A., 1931) state that there is no evidence that the sympathetic effects are aroused except through the central nervous system, and that there is no good reason for not believing that the afferent fibers belong to the dorsal roots and ganglia.

Davis and Pollock (Arch. of Neurology and Psychiatry, Feb., 1932), because of failure to relieve pain by sectioning of the posterior spinal roots and failure to produce analgesia in the de-afferented area, were led to the proposal that impulses could be conducted antidromically over the anterior roots. Failure of section of the posterior root of the fifth nerve in certain typical facial neuralgias to relieve pain also led to this same possible postulate. Their conclusions were reached experimentally by stimulating the superior cervical ganglion, after sectioning the anterior and posterior roots, separately and both of them, in conjunction, with and without sectioning the posterior root of the fifth nerve. Only sectioning the posterior spinal root and the posterior root of the fifth nerve at the same time prevented pain when the superior cervical ganglion was stimulated. Their conclusions were against accepting the above mentioned premises and that there were no antidromic sensory fibers in the cervical anterior roots and that the appreciation of pain is effected by a stimulation of efferent fibers which in turn stimulate the ordinary accepted sensory pathways in the cranial nerves.

H. Nelson and Chas. H. Frazier (Brain, March, 1932) investigated patients complaining of neuralgia after severance of the posterior root of the fifth nerve. They found that after a period of years the sensitivity to deep pressure increased one hundred fold, also that there was a pronounced improvement in point localizations and a better response to high temperatures. These were all lost when an accompanying thoracic sympathectomy had been performed. Their conclusions were inclined to agree with those who feel that the sympathetics and parasympathetics do contain afferent as well as efferent fibers.

Mixter and White (Arch. Neur. and Psych., May, 1931) conclude that it is difficult to prove that sensory impulses are carried by any but the cranial and spinal nerves in spite of some clinical evidence to the contrary. Pain-bearing fibers may run in many of the sympathetic nerves, but whether they connect with the sympathetic ganglia is not known.

Bishop and Heinbecker (Am. Jour. Physiology, May, 1932), in an analysis of the cervical sympathetic system consider, from their experimental work, that there is no support for the dictum of Gaskell's that preganglionic fibers are always myelinated and postganglionic fibers are always unmyelinated.

H. W. Woollard (Proceedings of the Royal Society of Medicine, Aug., 1932) states that there is no reason for discarding the opinion that all afferent fibers belong to the dorsal ganglia. Since there is no question as to the mixture of fibers in nerves, the function of medullated and nonmedullated fibers and as to just which system claims certain afferent fibers is not of such great clinical importance.

The endocrine system precedes the involuntary nervous system in evolutionary development. It is a balancing co-ordinating system with a diffusibility of action much as the sympathetc system. Cannon has called attention to the fact that the impulses from the sympathetics and from adrenin (from the medullary portion of the adrenals) not only have a similar effect on the tissues (the sweat glands being excepted) but have a reciprocal stimulation one for the other, one a chemical and the other a nerve impulse.

W. J. Mayo (Interstate Post. Grad. Assembly, N. A., 1929) emphasizes the close relationship between the sympathetic system and the suprarenals and the hypophysis and the logical reason for the same, since the latter are known to contain cells from the anlage of the nervous system.

The endocrines represent a concentration and specialization of chemical substances. The thyroid has a functional relationship to the sympathetic system somewhat similar to that of adrenin. The reciprocal relationship between the pituitary body and the sympathetic system has not been proven.

The parasympathetic system has been considered by some, but not proven, to hold a possibly similar position in relation to the glands of digestion and adnexia and to the parathyroids.

The fluctuating balance between the sympathetics and the parasympathetics, largely maintained through efferent impulses, coordinates cellular activities. The former is catabolic and is stimulated when excess energy is demanded. Fatigue brings about sleep, and during sleep with diminished afferent sensory impulses, there is a loss in sympathetic tonus. The parasympathetics are in a relative ascendency, without an actual increase in parasympathetic tonus, with a consequent restorative influence to body tissues.

H. P. Gilding (Jour. of Physiology, Jan., 1932) demonstrated the course of the sympathetic nerves as they follow the motor and sensory nerves to the periphery, branching from them in the vicinity of the minute blood vessels innervated.

Little is known of organic disease of the involuntary (interofective) system in contrast to well known morbid anatomy of the central nervous system. There are conditions in which one division is apparently overbalancing the other; an increased irritability of the neurones of one division over the other. In one who is vagotonic or parasympatheticotonic there is generally a slow pulse, respiratory arrhythmia, hypotension, hypoglycemia, increased glandular activity, including the bronchi, lacrimal and salivary glands, hypermotility of the gastro-intestinal tract, with possible spastic constipation and colitis and vomiting.

In one who is sympatheticotonic, the heart is apt to be accelerated and there is an undue disturbance of appetite and digestion, dilation of the pupil and exophthalmos from emotional causes. Abnormal raising of the blood pressure may occur with physical and emotional stress.

In the sympatheticotonic, adrenin has an undue effect in increasing the pulse and systolic pressure. There are other tests, as Lowi's pupillary test, that of dermographia, Pende's pilomotor reflex and Ruggeri's test, that of the acceleration of the pulse by extreme convergence. There is also the cold, clammy skin, lack of salivary and lacrimal secretion, as well as decreased mucous

secretion in the nose and throat. High blood pressure in women is sometimes due to sympathetic irritation.

Vagotonia and sympatheticotonia are relative terms. While there may be an apparent increase of functional activity of one division over the other, the real status may be due to the depressed activity of one division rather than an actual hyperactivity of the other. Eppinger and Hess stated that heightened activity in one division precluded the same condition in the other. Petren and Thorling maintained the reactions at times manifested to certain drugs could only be explained by assuming a co-existing increased irritability of both divisions. There is no general accord for many of the symptom complexes associated at times with diseases relative to the dominating division of the involuntary system.

Tuberculosis in the second and third stages suggests a vagotonia, yet there may be the gastro-intestinal symptoms of sympatheticotonia. Hyperthroidism suggests symptoms akin to those where the sympathetics are dominant, yet there may be a hypermotility of the gastro-intestinal tract, as seen when the parasympathetics are in the ascendency.

Eppinger and Hess regarded eosinophilia as a symptom of vagotonia. It is not an exclusive attribute of parasympatheticotonia, occurring in certain skin diseases and intestinal parasitisms, but it has been stated that it never occurs in sympatheticotonia.

Experimental results are influenced by the individual, the dosage of drugs employed and the manner of their administration, and probably accounts for the diversity of opinions expressed. The rise in blood pressure is one of the most constant reactions to sympathetic stimulation. And this is profoundly influenced by the acid-base cellular content. With a high potassium-ion content, tending toward greater alkalinity, this is an increased sympathetic reaction and rise in blood pressure from administering adrenalin. The reverse is true when the blood shows a tendency to the acid side.

While a state of sympatheticotonia cannot be said to be a cause of hyperthyroidism, since it may be a result of this condition, yet it no doubt is an important factor. Likewise is the vagotonic state a factor in bronchial asthma. The factors that activate the two

divisions of the interofective system are far from being fully established.

The functional condition of the cardiovascular system is one of the best criteria of interofective balance. Toxemias are known to increase sympathetic tonus, and when actively present in a disease, as tuberculosis, vagotonic in type, there may be sympatheticotonic symptoms.

The clinical evaluation is not always easy. There is often a similarity between the endocrinopathies and irritability of the involuntary (interofective) systems. There is one difference to be borne in mind: the involuntary system does not persistently affect the basal metabolic rate.

The action of atropin is limited largely to the cranial division of the parasympathetics and has a paralyzing effect on its terminal filaments, vasodilation excepted. Hence the effect of atropin is much greater in those who are distinctly sympatheticotonic. Pilocarpin acts as a stimulant to the parasympathetic fibers, and particularly to its secretory fibers, and when they are markedly in the ascendency readily produces salivation. Vagotonia is commonly associated with hyperacidity of the tissues. We have no positive knowledge of any hormone that acts directly as a parasympathetic stimulant, but changes in the acid-base cellular content do seem to result in changes in the balances of interofective function.

There is a decided interdependence between the ionic cell content and the functioning of the involuntary nervous system. It is a question whether the allergies, particularly asthma and hay fever, primarily represent an anaphylactic condition to certain substances. The increase of cell permeability, its physical or electrolytic state, the increase of H ions or OH ions, the potassium and sodium content as against the calcium content, are possibly the more primary factors. The bronchi and lungs belong to the enteral system. The motor cells of the parasympathetics lie in the walls of the bronchi, the vagus supplying the connector fibers.

Sympathetic fibers course with the blood vessels and relax musculature and depress secretion. Serum disease, anaphylaxis, the urticarias, asthma, hay fever and bronchitis present more or

less the picture of parasympathetic hyperirritability. Anaphylaxis as an example of general stimulation of the parasympathetic reflexes will be recognized by an increased muscular and secretory activity.

The caliber of the blood vessels influences the volume of blood in the tissues. Their rhythmic contraction and tonus is essentially myogenic in nature, but not entirely free from regulatory nervous influences, hormones and other substances in the blood. The vasoconstrictor mechanism plays the dominant rôle in the nervous control of blood vessels. The sympathetic system acts principally as a vasoconstrictor. While the parasympathetic nerves no doubt do contain vasodilator fibers and there is at times a vasodilator reflex mechanism, yet we have no proof of a general distribution of vasodilator fibers. Vasodilation may accompany glandular activity, and as such is explained the flushing of the mucosa of the mouth and pharynx following stimulation of the cervical sympathetic trunk. Vasodilation and the relief of pain have undoubtedly followed periarterial sympathectomy. The fact that the arteries receive their sympathetic innervation at intervals from contiguous nerves makes difficult the establishment of any rational theory as to why relief is at times accomplished. The results as published certainly do not justify the indiscriminate application of this form of therapy, and it is without practical application in otolaryngology.

The regulation of the pulmonary circulation is largely mechanical and dependent in a large measure upon the control exercised over the systemic circulation. The vasomotor control of pulmonary vessels through the involuntary nervous system occupies a very subordinate position.

Carlson anl Lockhardt (Amer. J. Phys., 1921, 55:31-32) observed in frogs that sympathetic stimulation did not constrict pulmonary vessels, though they were so affecetd by adrenalin. They also observed that stimulation of the vagus did constrict pulmonary vessels. The neurologic regulatory influence over the blood supply in the tissues presents such differences in response, variations even to the point of reversed responses, due, no doubt, to other factors, so that clinical deductions as regards the sympa-

thetics and parasympathetics and the state of the blood supply are impossible.

Sympathetic ganglionectomy or ramisection is an attempt to produce complete sympathetic denervation. It received its greatest impulse towards popularity following the report of Jonnesco (1916) in attempting by sympathetic ganglionectomy to relieve anginal pain. Similar reports have been made in attempting to relieve bronchial asthma. Some successes through this procedure have undoubtedly been achieved, but they still remain strongly in the minority. Here again the procedure lacks rationale, since the sympathetics are held to be bronchodilators. Some have theorized that the resulting powerful parasympathetic preponderance causes a reversal of action on the bronchial musculature. Again some hold that the clinical successes are due to reduced afferent impulses.

The distribution of the leucocytes is affected by nervous control (F. Hoff, 1928, Die Vegetative Regulation des Blutes, Deutsches Wohnschr., 54:905-908). Other variations in the blood picture are closely correlated with manifestations of functional imbalances of the autonomic nervous system. The output of myelocytes by bone marrow is increased by experimental sympathetic stimulation and the lymphocytes by vagus stimulation. There are facts that support the theory that endothelial permeability is modified by autonomic nerve stimuli; that an overbalance towards the sympathetics tends to a leucopenia, and when towards the parasympathetics to a leucocytosis. There is also experimental evidence that the initiation of immune reactions is initiated through nerve stimuli.

The histopathologic knowledge of the autonomic ganglia as related to disease is very scanty. Arteriosclerosis is accompanied by ganglion cell changes and by some considered to be a genetic factor.

Muscular dystrophies have been shown to have accompanying ganglion cell changes and this condition has followed sympathectomies. Imagawa reported dystrophic changes in the muscles of the tongue following section of the chorda tympani.

Psychic processes are not limited to the cerebral cortex. One's emotional life may be profoundly affected by the balances between

the two divisions of the interofective nervous system and, to reverse the process, visceral dysfunction may be due to emotional disturbances.

The function of the cranial division of the parasympathetics consists largely of a group of reflexes, motor, sensory, trophic and secretory, that are conservative, protective and upbuilding. Asthma produced reflexly through pressure in the nose may be tested by cocainization of the nasal mucous membranes. No trophic nerves have ever been demonstrated, and the nervous control over cellular nutrition can more logically be conceived as a balancing of efferent impulses. The afferent fibers of the tenth have a relationship to its own motor neurones and to the sensory and motor neurones of the other cranial nerves similar to that occupied by the dorsal spinal nerve roots to the sympathetic fibers. Afferent fibers of the fifth nerve have an analagous reflex relationship to the seventh, ninth and tenth nerves. As a rule, the sensitivity of a mucous membrane decreases in proportion to its distance from the natural opening to the exterior. This is not true of the larynx, which possesses an extreme sensitivity and serves as a very important protective mechanism. This may account for the relatively large size of the superior laryngeal nerve. The larynx serves as a barrier to foreign bodies and, when necessity demands, the afferent impulses are sent through this nerve. The reflex act results in not only closing the larynx but in exciting a reflex cough which serves as an expelling force. In addition, the act of swallowing is reflexly stimulated. In the larynx the early hoarseness expressed reflexly through the superior and inferior laryngeal nerves becomes an important sign in tuberculosis.

The parasympathetic trophic reflex is seen in an atrophy of laryngeal tissues in pulmonary tuberculosis. The afferent and efferent impulses both travel in the vagus, thereby establishing a rational explanation for the laryngeal involvement being more often on the side of the pulmonary lesion. Sensory reflexes leading to irritation and cough may come from the parasympathetics in the lungs, pleura, larynx, tonsils and from the external auditory canal via its branch from the vagus.

Swallowing is a reflex act, partly voluntary and partly involuntary. The upper one-third of the esophagus contains striated

muscle and the lower two-thirds unstriated muscle. It has been
taught that the esophagus was without sympathetic innervation
but this is probably not true.

Carlson and Lockhardt demonstrated (Am. J. Phys., 1921, p.
299) the presence of sympathetic fibers in the lower third of the
esophagus. The sensory reflex from cold, that of brow pain, dem-
onstrates the relationship between the tenth and the fifth nerves.
It is a similar relationship to that between the sympathetics and
the spinal sensory nerves.

The salivary glands receive a dual involuntary innervation.
The submaxillary and lingual glands receive their sympathetic
impulses through the external maxillary plexuses, and the para-
sympathetic through the chorda tympani of the seventh; the par-
otid, their sympathetic innervation through the middle meningeal
plexuses and their parasympathetics through Jacobson's nerve of
the ninth. The sympathetics decrease the watery and salt ele-
ments and stimulate the organic contents of the saliva. The
reverse is true of the parasympathetics. There is the known influ-
ence of the sense of sight, smell and mental ideas, but essentially
the glands functionate through the involuntary nervous system.
The vagotonic is more apt to suffer from an abnormal amount
of saliva. The sympatheticotonic will have more or less of a dry
mouth, as particularly exemplified during extreme sympathetic
stimulation through fear and anger. Sympathetic paralysis or
operations upon the sympathetics may be followed by an increase
in the flow of saliva, demonstrating a predominating influence
of the parasympathetics.

Excessive secretion in the upper respiratory tract may result
reflexly from pulmonary and gastro-intestinal disorders. It has
been a popular lay conception that so-called catarrhal conditions
precede tuberculosis as a causative factor, while it is probably
nearer the truth that the condition is a reflex disturbance from the
pulmonary lesion.

The mucous membrane of the nose, accessory sinuses, buccal
cavity and pharynx receive a double involuntary innervation, the
fibers following those of the fifth from the sphenopalatine gang-
lion, the sympathetics originating through the deep petrosal and
the parasympathetics through the seventh nerve. In the pharynx

the fibers of the ninth serve in a similar capacity to those of the seventh.

The dryness in the nose in fever is not so much the direct result of the fever as it is a toxic stimulation of the sympathetics, resulting in a constriction of blood vessels and lack of glandular secretion.

Deafness has proven a most unsatisfactory therapeutic problem. The pathology of otosclerosis presents trophic changes of bone cells. The diagnostic differentiation of true otosclerosis from other types of deafness is far from satisfactory. The vulnerability of the eighth nerve is well established. Anatomically the involuntary nervous system supplies a plexus, the tympanic plexus located on the medial wall of the middle ear. It receives its sympathetic innervation through the caroticotympanic nerve from the internal carotid plexus and its parasympathetic innervation by a tympanic branch from the petrosal ganglion. If one may be permitted to theorize, the extracellular control of cellular activities offers logical reasons for being a factor in certain types of deafness. Psychic or emotional deafness may be due to a disturbance of such extracellular correlating agencies. While clinical evidence as regards the involuntary (interofective) nervous system is not so readily demonstrated, we do have ample evidence as to the influence of the chemical or endocrine correlating system. Deafness, progressive in type, demands an investigation of the patient's basal metabolism. Deafness with hypothyroidism offers one of the most striking instances of improvement through endocrine therapy. Since there is a synergism and even some parallelism between the thyroid secretion and the sympathetic nervous system, it is not assuming too much to hope that further knowledge as regards the imbalances of the sympathetic and parasympathetic systems will lead to additional help to those afflicted with certain types of deafness.

A. B. Murphy, in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (March, 1933, p. 166), comments on the lack of uniformity in the results reported in the experimental work that has been done as regards the effects of variations in vascularity on the functions of the ear. Vasodynamic drugs, particularly those that affect the interofective nervous system, have been used

experimentally. A. B. Murphy, in presenting his thesis, found that after interrupting cervical sympathetic innervation, however, there were no spontaneous phenomena relative to the internal ear. He suggested that the vasodilatory mechanism of the internal ear may be primarily a part of the cranio-autonomic system, and that the knowledge of the influence of the sympathetic system offers little in the way of positive deductions.

The involuntary nervous system, in carrying motor and trophic impulses, also to a modified degree carries pain-bearing sensory impulses. To fully develop the sensation of pain there is added the co-operation of the fifth and the spinal sensory nerves. Pain through the involuntary system requires a marked disturbance of the tissues. It is as if, after a continued bombardment of stimuli not in themselves sufficient to be recognized, there develops a neurone hyperirritability. This is seen in the toxemias of various diseases, where though the toxin does attack the cell directly it also has an important effect through the involuntary nervous system. Referred pain from the viscera of the body follows certain segmental laws. This is due to the fact that efferent and afferent stimuli, as regards any particular part, start from and go back to the same segment of the central nervous system. In the midbrain and bulb there is a similar proximity of neurones which permits this same transference as from the vagus to the fifth nerve. It is this same intimate relationship that permits of sinus pain to be transferred to other localities than the particular part involved.

CONCLUSIONS.

The cells of the body possess certain inherent powers of activity based upon their colloid and crystalline content, following certain physical and chemical laws. The co-ordinating of cellular activities depends upon the actions of the chemical substances of the endocrine system and upon nerve impulses through the involuntary (interofective) system. The otolaryngologic field is but a part of the whole. It is likewise under the influence of these co-ordinating forces. The co-ordinating nerve impulses, when balanced one against the other, materially assist in maintaining the rhythms of life. They are forces that have a definite place as factors in maintaining the so-called resistance of the body. They

are part of what is generally a dual system, with the two components commonly opposing each other. Since there is clinical evidence at times of the predominance of one division of the involuntary (interofective) system over the other, the recognition and consideration of these imbalances in the study of disease are worthy of consideration.

The known anatomy and physiology of the involuntary (interofective) system are far from complete. The initiation of nerve impulses and the manner in which these impulses affect body cells has not been fully proven. The complexities of the chemistry of body metabolism obscure and make difficult a definite association of symptomatology with the involuntary (interofective) nervous system. In its present development clinical application is disappointing but seems to possess future possibilities.

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REFERENCES.

Pottinger, F. M.: Symptoms of Visceral Disease.
Kuntz, Albert: Autonomic Nervous System.
Cannon, W. B.: The Wisdom of the Body.
Cannon, W. B.: The Linacre Lecture on the Autonomic Nervous System.
Marriott, W. McKim: Recent Advances in Chemistry in Relation to Medical Practice.

XII.

METHODS FOR PREPARING AND STUDYING TEM-
PORAL BONE SPECIMENS.*

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During the past year, in the Department of Otolaryngology, State University of Iowa, specimens of the temporal bone suspended in liquid have been studied with the binocular dissecting microscope and strong illuminating lamp. By this method more details may be observed than by gross observation, and also depth and perspective are obtained which are not possible with the flat microscopic section.

The studies to date have been chiefly made upon whole and dissected specimens of the infant's temporal bone, but sufficient work has been done with the adult bone to make it seem practical also in this field. Because of the many requests for the technic of preparing and mounting these specimens the following procedures are offered.

I. Preparation of the infant's temporal bone to show normal structures of tympanum and antrum :

(Where there has been congestion of the circulation of the head before death, as in birth injury or craniotomy, the blood vessels of the drum and tympanum may be well shown.)

1. Remove complete temporal bones intact from cadaver and fix in 10 per cent formalin for two to three months or longer.

2. Wash with running water—forty-eight hours.

3. Remove excess soft tissue and make dissection with aid of dissecting microscope.

4. Dry in air twenty-four hours.

5. Mount in liquid petrolatum.

BLOOD SUPPLY OF INFANT DRUM
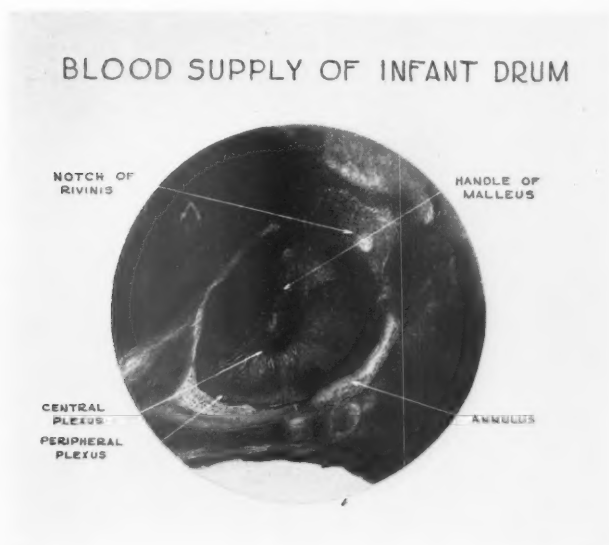
Fig. 1.
Figs. 1 and 2 are drawings to show type of specimens prepared
by the first method.

II. Preparation of infant's temporal bone for transparency of drum and some transparency of bone:

1, 2 and 3. Same as above procedure.

4. Dehydrate by suspending specimens in the following solutions:

80 per cent alcohol; 24 hours

95 per cent alcohol—No. 1; 24 hours

95 per cent alcohol—No. 2; 24 hours

Absolute alcohol—No. 1; 24 hours

Absolute alcohol—No. 2; 24 hours

Absolute alcohol and ether (equal parts)—No. 1; 24 hours

Absolute alcohol and ether (equal parts)—No. 2; 24 hours

Blot and dry.

5. Mount in cedar wood oil.

VIEW FROM ABOVE DOWN INTO
TYMPANUM

HEAD OF MALLEUS

CHORDA
TYMPANI

BONY CANAL
FOR TENSOR
TYMPANI
MUSCLE

LONG HANDLE
OF MALLEUS
ATTACHED
TO DRUM

TENSOR
TYMPANI
MUSCLE

STAPEDIUS
TENDON

PYRAMID

COCHLEARI-
FORM
PROCESS

STAPES

VESTIBULE
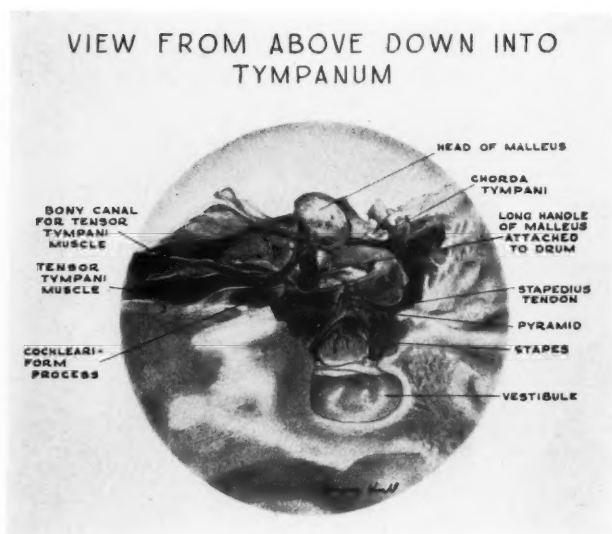
Fig. 2.

III. Preparation of adult specimens:

Adult specimens may also be prepared as in I and II, but for ease of dissection, and particularly for cross-section specimens, the standard method of preparing the temporal bone for microscopic sections (used at various laboratories) is followed. In this way, also, both adult and infant temporal bones may be made quite transparent for demonstration of the labyrinth.

The method is as follows:

1. Fix specimen in 10 per cent formalin; 14 days or longer
2. Wash in running tap water; 48 hours
3. Zenker's solution; 24 hours
4. Wash; 24 hours
5. 80 per cent alcohol; 24 hours
6. 95 per cent alcohol; 24 hours
7. 5 per cent nitric acid; suspend specimen until decalcified
8. Wash—running tap water; 48 hours
9. 5 per cent sodium sulphate—No. 1; 24 hours
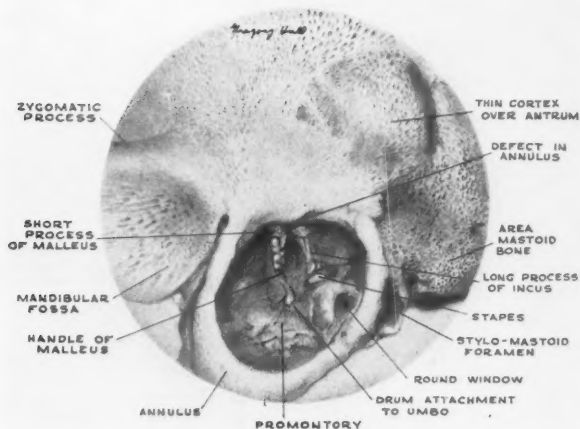
Fig. 3. Example of specimen prepared by second method.

10. 5 per cent sodium sulphate—No. 2; 24 hours
11. Wash—running tap water; 48 hours
12. Dehydrate—suspend specimen
    80 per cent alcohol; 24 hours
    95 per cent alcohol—No. 1; 24 hours
    95 per cent alcohol—No. 2; 24 hours
    Absolute alcohol—No. 1; 24 hours
    Absolute alcohol—No. 2; 24 hours
    Absolute alcohol and ether (equal parts) No. 1; 24 hours
    Absolute alcohol and ether (equal parts) No. 2; 24 hours
13. Celloidin
    2 per cent; 1 week
    4 per cent; 1 week
    6 per cent; 1 week
    8 per cent; 1 week
    10 per cent; 1 week
    15 per cent; 1 week

HORIZONTAL SECTION THRU
TEMPORAL BONE SHOWING
RELATIONS OF MIDDLE AND INNER EAR
(ADULT)
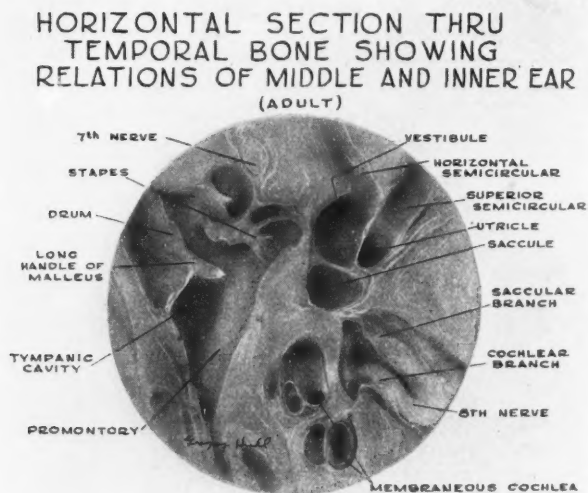
Fig. 4. Adult bone specimen (cross-section).

14. Solidify and section or dissect.

15. Mount in 70 per cent alcohol or liquid petrolatum.

When preparing either adult or infant specimens to show labyrinth, omit 13 and 14 above and, instead of mounting in oil, inject mercury with hypodermic needle through superior semicircular canal until labyrinth is filled and seal with transparent glue.

Lastly, suspend specimen in methyl salicylate or cedar wood oil for six months, or until the bone is quite transparent, and mount.

Any type of container having a smooth, transparent surface, such as glass or cellulose compounds, may be used for mounting. Up to the present time the most satisfactory material has been a cellulose product* which is obtained in sheets and may readily be cut to the desired dimensions. A convenient size is 1¾ inches square and 1 inch deep. The edges of the container are sealed, as well as the specimen fixed to the base, by means of a

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*Pyroxylin—made by Dupont Viscoloid Co., Arlington, N. J.
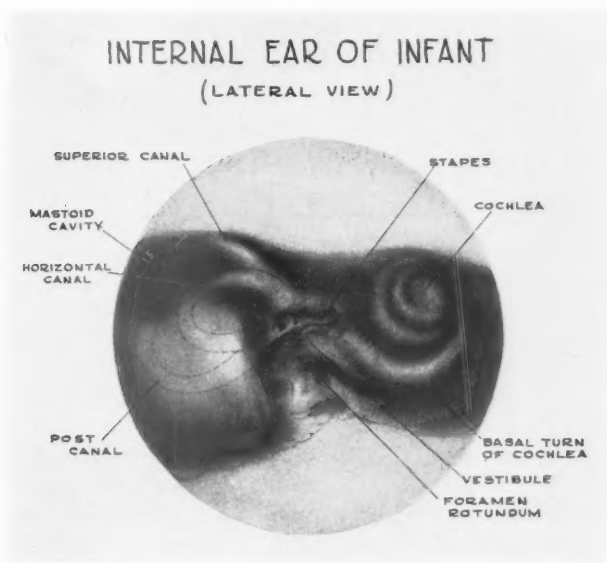
Fig. 5. Specimen with transparent bone and injected labyrinth.

glue made by dissolving small pieces of the cellulose product in acetone.

When the box is completed except for one end, the specimen is fixed in position and allowed to set for six hours. The container is then filled with oil to within one-fourth inch of the top and the final piece glued and held in place for six hours by a weight.

Lastly a small hole is bored in the bottom of the container and, by means of a hypodermic needle and syringe, the remaining air is replaced by oil. The opening is then sealed after first cleaning with chloroform or ether.

The disadvantage of a cellulose product is that it will become opaque and soft when in contact with methyl salycilate solution or alcohol over a period of time. Glass containers are necessary when the above liquids are used for mounting.

XIII.

THE EFFECT OF IRRADIATED VASELIN IN NASAL PATHOLOGY.*

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Because of the proven value of ultra-violet light as a powerful bactericide and because of its irrefutably tonic and healing effect when applied to cutaneous lesions, it was decided to try its effect upon nasal conditions, such as chronic ethmoiditis, vasomotor rhinitis, atrophic rhinitis and polypoid degenerations. But because of unsatisfactory results which we thought were due partly to the mechanical impossibility of getting the ray behind polyps, turbinates or hyperplasias, and because of its lack of penetration into the sinuses, which are the chief sources of the visible pathology and because of the difficulty in getting the patient to constantly return to the office for irradiation, we decided to use irradiated vaselin as a substitute.

This had several practical advantages: (1) The patient could instil it several times a day if necessary at home; (2) it could be injected into the sphenoid or maxillary sinus or by the method of Proetz be sucked into the ethmoids.

E. H. Eising[1] and F. S. French[2] have probably contributed most to our knowledge of the nature of irradiated vaselin. The following is a brief summary of their results:

1. Exposure of petrolatum to ultra-violet irradiation increases its acidity.

2. Creates sterols.

3. Turns it darker in color.

4. Gives it a photochemical action.

5. Gives it a bactericidal power.

This last has been further substantiated by Thompson and Sheard[3] and A. Treloar Ross of the University of Oregon.[4] Ross has shown that vaselin which has been exposed to ultra-violet

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*From the Department of Otology, University of Oregon Medical School.

light for two hours will, when mixed with cultures of staphylo-
coccus aureus and pyocyamus, render the mixture sterile within
twenty-four hours. He states further that the effect is bacteri-
cidal rather than bacteristatic.

Eising[3] reports excellent results in eighty-four cases of abdomi-
nal wall suppurations, one granuloma inguinale, eighteen of
chronic osteomyelitis and others such as tenovaginitis, gas bacillus
infection, empyema, carbuncle of the neck, varicose and diabetic
ulcers.

French[2] makes the following statement: "Ultra-violet irradi-
ated petrolatum has the property of emitting an energy which
can be detected and roughly measured as to intensity by means
of photographic plates. This energy is radiant in character and
not chemical. It is not ultra-violet light but an as yet unidentified
form of radiant energy which is transmitted by celluloid but not
by quartz or glass. . . . In petrolatum the radiant activity can
be separated by mechanical means and obtained in the form of a
dry amorphous powder which is itself strongly radiant."

The present work reported is a summary of the clinical results
with irradiated petrolatum used in the treatment of 100 cases of
infection of the nose and accessory sinuses. These cases were
divided into two groups: First, those in which the patient was
given bottles of irradiated oil to instil in the nose three times a
day. Second, those in which the oil was injected into the sphenoid
or maxillary sinuses.

White vaselin was exposed to the rays from a mercury arc at
a twelve-inch distance for six hours. The depth of the layer of
oil under irradiation was one-eighth inch and the lamp tested with
a Burt photometer at 90 per cent. The irradiated petrolatum was
then diluted with an equal part of liquid petrolatum, which gave
it a melting point of approximately 95 degrees Fahrenheit.

The patients in the first group were instructed to lie down
with the head thrown backward far enough so that the oil would
remain in the upper part of the nose for at least fifteen minutes.
In this group both acute and chronic cases were treated. The
acute cases showed no definite clinical signs that would indicate a
more rapid recovery or a retardation of the condition. The
chronic cases, such as chronic rhinitis, ethmoiditis, those with

polypoid changes, and various allergies, showed no improvement. Many of the patients failed to continue treatment over as long a time as directed. However, by careful checking, we found twenty cases who followed directions and used the oil for at least one month. Four cases of atrophic rhinitis with crusting showed some improvement. Whether this was due to the lubricant effect of the oil alone or to the irradiation factor or both has not been determined, since controls have not yet been carried out. It is our opinion that any oil would have the same effect.

In the second series the oil was injected into the sinuses. Five cases of chronic maxillary sinusitis and two of chronic sphenoiditis were treated. None of these showed any definite clinical improvement. Two of these cases, No. 207 and No. 208, were of especial interest. They were both young adults with bilateral maxillary infection. The antra were washed and then filled with irradiated oil twice a week for a period of one month. Washings at the end of this period showed no macroscopic diminution in the amount of pus from the sinuses. At the end of the month radical surgery was done and the lining membranes were removed and sectioned for microscopic examination. This work was done as part of a research authorized by the American Academy of Ophthalmology and Otolaryngology. The report of these examinations made by Dr. Olof Larsell follows:

"No. 207. The epithelium shows considerable injury; cilia are present only in patches and then not normal in appearance. There are a few goblet cells and glands are present beneath the epithelium. Also some cyst-like masses of mucus are present. The tunica propria is very vascular and injected. There is marked fibrosis. There are follicle-like groups of lymphocytes and a diffuse scattering of lymphocytes. Numerous plasma cells are seen, but they are diffused beneath the epithelial surface. There are some atypical eosinophiles. Hemorrhage is seen in the deeper parts and also heavy bands of fibrous tissue.

"No. 208. There is considerable destruction of the epithelium and the cilia are largely destroyed. There are numerous goblet cells, and folds of epithelium extend into the underlying tunica propria. There are also tubulo-alveolar glands present. The outer portion of the tunica propria is very edematous with large spaces
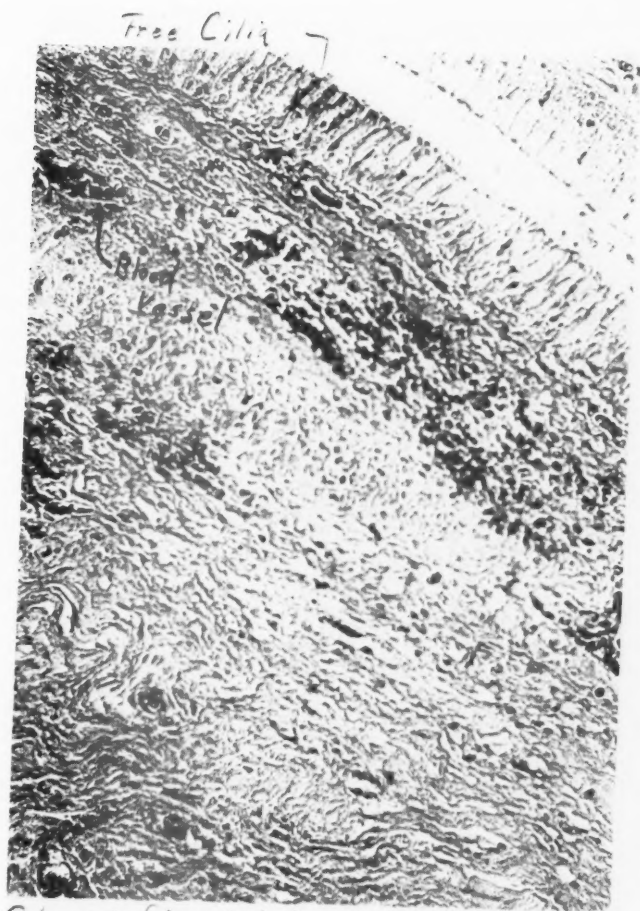
Chronic Sinusitis. #1. Untreated

Chronic Sinusitis Not Treated
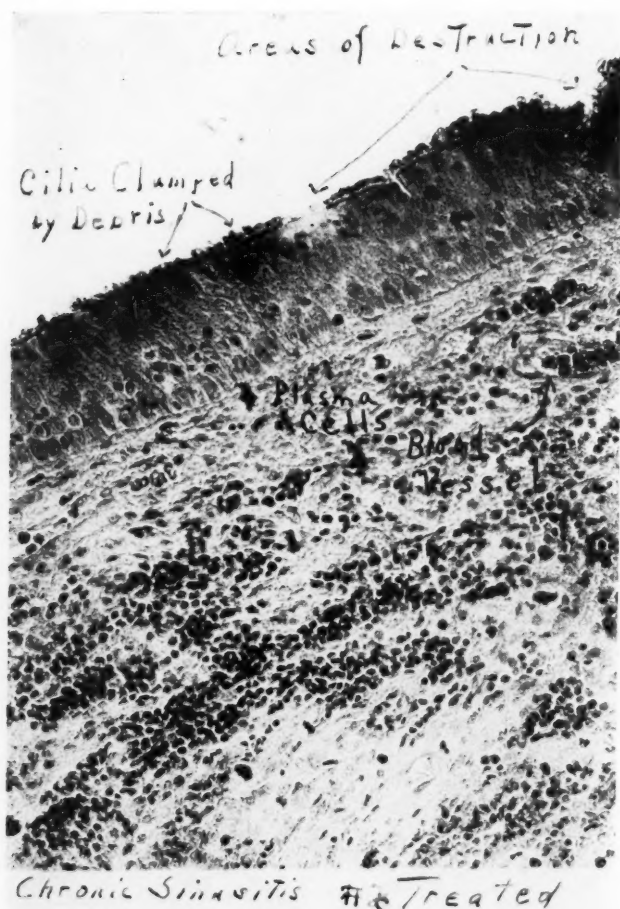
filled with fluid. There is a diffuse scattering of plasma cells and
lymphocytes. The latter are numerous and seen chiefly around the
blood vessels. In the deeper tissue there is considerable fibrosis."

For the sake of comparison a control slide showing a section of
the membrane from a case of chronic sinusitis not treated with oil
is presented. In this it will be noticed that the cilia show much less

destruction and do not seem to be so clumped nor to contain so much débris. Otherwise the same chronic inflammatory changes are seen to be similar in both.

It is interesting to note that Noah Fox[6] reports a similar effect on the ciliated epithelium from the noses of rabbits which had been sprayed with plain liquid petrolatum daily for nine months. He states that microscopic sections of these membranes show: "The surface epithelium is desquamated, and many polymorphonuclear leucocytes and plasma cells are present. There are a few polymorphonuclears in the substratum. There is a tendency toward new growth of the epithelium."

CONCLUSIONS.

1. Irradiated vaselin is of no clinical value in the treatment of the nasal membrane. The harmful effect which it produces is probably due to the oil itself and not the radiant element in the oil.

2. These results have no bearing on the effect of ultra-violet light in the nose, as the stored up energy in the vaselin is not ultra-violet light.

3. The bactericidal power of the irradiated oil used was not sufficient to sterilize the nasal mucous membrane.

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REFERENCES.

1. Eising, E. H.: Ultraviolet Irradiated Petrolatum, Its Physiochemistry and Bacteriology. Medical Journal and Record, 136:251-253, September 21, 1932.

2. French, F. S.: Ultraviolet Irradiated Petrolatum. Clifton M. Bull, 18:21-34, April, 1932.

3. Thompson, L., and Sheard, C.: The Bactericidal Action of Irradiated Oils. Proceedings of the Staff. Mayo Clinic, 6:690-691, November 13, 1931.

4. Ross, A. T.: Bactericidal Property of Ultra-violet Irradiated Petrolatum-Lanolin mixture. Proc. Soc. Exper. Biol. and Med., 29:1265-1267, June, 1932.

5. Eising, E. H.: Clinical Observations in the Use of Ultraviolet Irradiated Petrolatum. American Journal of Surgery, 19:244:249, February, 1933.

6. Fox, Noah: The Effect of Camphor, Eucalyptol and Menthol on the Nasal Mucosa. Arch. Otolaryng., 11:48-54, January, 1930.

TRACHEOTOMY: A STUDY OF 200 CONSECUTIVE CASES.*

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Tracheotomy, one of the oldest of surgical procedures, still remains one of the most useful. The indications for, and the technic of, the operation are so well established that little new information at present can be added to the already voluminous literature on the subject. However, the detailed review of a group of these cases, reported herewith, serves to emphasize a number of points of practical value.

The 206 consecutive tracheotomies under consideration were carried out on 200 patients in The Mayo Clinic from January 1, 1929, to and including the first six months of 1933. The series consists of 174 operations performed on the laryngologic service, and thirty-two performed on the general surgical service. Seventy-one operations (34 per cent) were performed as emergency measures in order to relieve impending suffocation, although a degree of respiratory difficulty was present before in 102 of them. One hundred thirty-five operations (65 per cent) were carried out as a matter of expediency, either to lessen the risk of a radical laryngeal operation or to obviate impending respiratory obstruction.

The patients ranged in age from less than a year to more than 75 years. Each of the two youngest patients in the group was 8 months of age. Both of them were males and had foreign bodies in the trachea. The two oldest patients in the series each was 76 years of age. One was a man; the other, a woman; both had carcinoma of the larynx. One hundred twenty-five patients were more than 50 years of age, and 153 more than 40 years. Seven-

teen were less than 5 years of age; twelve had foreign bodies, three had multiple papillomas and two had laryngotracheitis.

The males in the series numbered 166, the females, thirty-four. This preponderance of males over females is due chiefly to the large number of cases of laryngeal carcinoma in the group. As is well known, carcinoma of the larynx is very much more prevalent among males than females. Among the 133 patients with laryngeal malignancy included, there were 128 males and five females. In the cases of multiple papilloma of the larynx and of laryngotracheitis the males likewise outnumbered the females; in the former group the proportion was 5 to 3 and in the latter, 4 to 1. The group of patients with foreign bodies contained an equal number of males and females. Only among patients who had goiter did the number of females exceed the number of males; fifteen were females and ten, males.

The primary pathologic conditions necessitating tracheotomy in the series included a wide variety of lesions. The most frequently encountered of these was carcinoma of the larynx, which was present in 133 cases. In several of these, tracheotomy was performed on two different occasions because of recurrence of the malignant process. In twenty-five cases pathologic change of the thyroid gland was either directly or indirectly responsible. In many of these the condition was malignant. Fourteen patients had foreign bodies in the tracheobronchial tree, and eight had multiple papillomas of the larynx. There were two cases each of amyloid tumor of the larynx, and of gumma of the larynx. In two cases both vocal cords were in the median line, secondary to carcinoma of the esophagus. A primary carcinoma of the trachea was responsible for respiratory obstruction in one case, and a metastatic carcinoma of the trachea secondary to carcinoma of the lower lip in another. Acute streptococcal laryngotracheitis was present in four cases, and chronic subglottic laryngitis in one. The last condition improved to a marked extent following tracheotomy, and the cannula was removed after four months. About two and a half months later it became necessary again to open the trachea in emergency resulting from recrudescence of the inflammatory process. Extensive cellulitis of the neck, with deep-seated abscess, was present in one case. Myasthenia gravis affect-

ing a man 47 years of age had resulted in such marked relaxation of the tissues of the hypopharynx, and of those about the larynx, that the pendulous epiglottis tipped posteriorly and inferiorly to such an extent that at intervals it blocked the superior laryngeal aperture, producing temporary obstruction.

A man, 59 years of age, came with such marked cicatricial stenosis in the subglottic region, secondary to emergency tracheotomy in childhood, that it was necessary to open the trachea immediately. Judging from the history, the original tracheotomy apparently had been done to relieve obstruction caused by diphtheria. Although the patient had been short of breath on exertion since childhood, acute obstruction had developed only shortly before he came to the clinic, and appeared to have resulted from increased crusting about the stenosed lumen, together with ulceration secondary to this. Following tracheotomy the scar tissue was dissected out through laryngofissure and the lining restored by means of a Thiersch skin graft after the method of Arbuckle. The tracheal cannula was removed fifty-four days later.

An infected, thrombosed hemangioma of the larynx was removed through laryngofissure, and tracheotomy was done at the same time because of persistent oozing. In another case tracheotomy became necessary because of perichondritis following electrocoagulation of a prolapsed ventricle.

Edema of the hypopharynx and larynx of indeterminate etiology, but associated with pericarditis with effusion and pulmonary infarct, necessitated tracheotomy in the case of a man of 35 years.

A man, 66 years of age, underwent block dissection of the cervical lymph nodes. Ten days later, during induction of anesthesia to permit coagulating an extensive epithelioma of the base of the tongue, acute obstruction developed. The trachea was opened immediately and the patient was resuscitated by artificial respiration.

Various pathologic conditions of the thyroid gland, and complications associated with thyroidectomy at times necessitate tracheotomy. These cases may be divided into several distinct groups: (1) Those in which obstruction occurs entirely independently of surgery; (2) those in which obstruction takes place at the time

of thyroidectomy; (3) those in which respiratory difficulty develops a few hours after operation, and (4) those in which dyspnea does not come on until several months after thyroidectomy. In the first group, either because of inflammatory or neoplastic involvement of the thyroid gland, there is encroachment on the tracheal lumen from compression, displacement, invasion or edema. Obstruction occurring during the course of thyroidectomy results either from direct surgical trauma to the recurrent laryngeal nerves or from tracheal collapse. Respiratory difficulty developing within a few hours to a day or two after removal of the thyroid gland may result from collapse of the trachea, from hemorrhage into the operative wound, from edema of the larynx or trachea or from paralysis of the vocal cords brought on by postoperative inflammatory reaction about the recurrent laryngeal nerves. In cases in which paralysis of the vocal cords develops shortly after operation, the loss of function usually occurs about twenty-four hours following operation and is likely to be gradual in onset, but may be abrupt. Return of function is to be anticipated in such cases, although it may not be complete, and in rare instances fixation is probably permanent. Dyspnea which comes on several months after thyroidectomy is the result of paralysis of the vocal cords which occurred at the time of operation. Immediately following injury in cases of this sort the vocal cords assume the cadaveric position and the patient is accordingly aphonic but has no respiratory difficulty. In the course of a few days to a few months the vocal cords gradually draw to the middle line, and the voice returns, but dyspnea of varying degree develops. Such fixation is invariably permanent.

Some degree of respiratory difficulty was present in 107 of the cases in the series. It is obviously impossible accurately to compare the severity and seriousness of this as it affects different persons, since a number of other factors, aside from reduction in caliber of the affected respiratory passage, are concerned. Among these are the age, general physical condition and vitality of the individual, the presence or absence of an acute infectious process or of a metabolic disturbance, and the duration and nature of the obstruction. However, an attempt has been made to compare the degree of dyspnea of the different patients by grading it 1, 2, 3

or 4, depending on the apparent seriousness of the clinical picture presented. The difficulty encountered in this regard can readily be appreciated when it is recalled that the degree of cyanosis is by no means an accurate criterion of the seriousness of the situation, since cyanosis is likely to be more pronounced in the presence of chronic obstruction, yet less threatening to the life of the individual. According to the scale presented, the respiratory difficulty in twelve cases was graded in 1; in twenty-five, 2; in fifty-four, 3, and in sixteen, 4. In the cases in which the difficulty was graded 1, dyspnea was noticeable only on slight exertion, while in those in which difficulty was graded 4 the patients were in extremis, and suffocation was imminent. Dyspnea graded 2 and 3 was between these two extremes.

The duration of the period of respiratory difficulty varied from a few minutes to several years. It was shortest in the cases of goiter and in those in which a foreign body was present, particularly in the former. Tracheal collapse developing at the time of thyroidectomy necessitated immediate tracheotomy in two cases. This condition occurred in one case when the anesthetic tube was removed from the trachea, after completion of thyroidectomy. Respiratory difficulty was of only a few hours' duration in most of the cases in which there were foreign bodies within the trachea or bronchi, although in one instance a safety pin apparently had been in the trachea of a child aged 13 months, and had produced dyspnea for two weeks. The longest periods of dyspnea were in the cases in which patients came to the clinic with bilateral paralysis of the vocal cords following previous thyroidectomy. In the three cases of this type respiratory obstruction had been present respectively for five months, two and a half years and four and two-thirds years. In one of these the dyspnea had developed at the time of thyroidectomy; in the remaining two cases it had appeared respectively five and six months after operation.

There had been no respiratory difficulty in ninety-five (71 per cent) of the 133 cases of carcinoma of the larynx. The period of dyspnea of those patients with malignant tumors of the larynx who presented evidence of obstruction at the time of the original examination varied from ten days to six months. One patient with an epithelioma of the larynx graded 3 had had some respiratory

difficulty for six months. Following preliminary tracheotomy laryngectomy was done and the patient recovered.

The great majority of operations were performed under local anesthesia; infiltration with procain was used in all of these. In fourteen cases no anesthetic was employed because of the existing emergency. In addition to the eight cases of goiter, in all of which an opening was made through the wound of the recent thyroidectomy, this group included two cases of foreign body, two of acute laryngotracheitis, and one each of multiple papilloma of the larynx and of acute phlegmon of the neck. Ether was given to one child, aged 13 months, who came with a safety pin in the trachea and had had moderate dyspnea for two weeks. A gas was used for anesthesia in four cases in which dyspnea developed following removal of foreign bodies from the air passages. All of these four patients were three years of age or less.

Unless a bronchoscope is introduced before tracheotomy, small children, as well as adults, are much more safely operated on under local anesthesia. During induction of general anesthesia of patients with moderate obstruction of the respiratory tract, increased mucoid secretion or spasm of the glottis may result in complete blocking of the air passage. The chief hazard in the use of a general anesthetic in such cases, however, is the possibility of relaxation of the accessory muscles of respiration, and of the pharyngeal muscles, resulting in complete obstruction. Even small children usually will co-operate sufficiently that it is possible to open the trachea satisfactorily, using local infiltration only. In this group, eleven patients less than the age of 10 years were operated on in this manner.

Injection into the tracheal lumen of a few drops of 10 per cent solution of cocain by introducing the needle of a hypodermic syringe through the membrane between two of the rings before opening the trachea, as suggested by St. Clair Thomson, was practiced as a routine. This does away with the coughing and struggling otherwise present and permits completion of the operation in a quiet, orderly manner.

In practically all of the cases in the series in which treatment was given in the Section on Laryngology, opening was made through an incision in the median line. In a few of the cases in

which general surgeons operated, the trachea was exposed through a transverse incision. Except in the unusual case, in which a neoplasm or other pathologic process overlies the trachea, incision in the median line is greatly to be preferred because of the ease of the procedure and its greater safety, particularly from the standpoint of hemorrhage. In one instance an extensive carcinoma originating within the larynx was perforating the thyroid cartilage and the overlying skin and infiltrating down over the upper portion of the trachea. A transverse incision just above the suprasternal notch permitted of opening the trachea to relieve the respiratory difficulty without breaking through into the neoplasm. In another case a carcinoma of the thyroid gland was encroaching on the upper portion of the trachea, producing marked dyspnea, and a transverse incision was employed to advantage. By incising directly in the median line of the neck, very little bleeding is encountered. In most instances the isthmus of the thyroid gland was divided rather than being displaced. The pressure of the displaced thyroid isthmus on the tracheal tube at times has appeared to increase the irritation within the trachea.

In most of the cases the trachea was opened by removing a circular disc of cartilage from one of the rings or from the lower border of the cricoid cartilage. Immobilizing the cricoid cartilage by means of small sharp hooks applied to its lower border greatly facilitates opening the trachea, especially if a patient has a short, thick neck. In a few instances a vertical incision was made through one or two tracheal rings, and a loop of silk suture was introduced through each end of one of the divided rings to permit of retraction in case the tube slipped out of the tracheal lumen, also, to serve as a guide for its replacement. Removal of a disc of cartilage has proved a much more satisfactory and safer procedure, for the opening does not tend to close as promptly in case the tracheal tube is displaced, and at the time of operation introduction of the cannula is more easily accomplished and there is less trauma to the trachea. In addition, there has been less local reaction postoperatively.

It is essential to provide ample drainage in all cases in order to guard against emphysema. A loosely rolled rubber tissue, a split tube or a Penrose drain placed just below the tracheal opening

and fixed with a suture to guard against its displacement will, as a rule, suffice. If the patient is coughing a good deal or if acute infection is present, it is often preferable to pack the wound loosely with iodoform gauze and leave it open. Healing is, as a rule, delayed very little, if any, in such cases, and there is surprisingly little infection of the soft tissues.

The optimal level for the tracheal opening varies in different cases, depending on the nature and extent of the primary pathologic process. Generally speaking, low tracheotomy is preferable in all cases except those in which the trachea is opened as a preliminary or intermediary stage in the removal of laryngeal carcinoma. The inferior extent of such a lesion will determine largely the level of the opening in such cases, since it is essential to avoid trauma to the tumor. At the same time a high opening facilitates laryngectomy. When possible, the respiratory tract in such cases is opened through, or above, the first tracheal ring. The level of the opening was noted in ninety-five of the 111 cases of laryngeal carcinoma in which the condition was operable. In nineteen of these it was made through the cricothyroid membrane, through or immediately below the cricoid cartilage in forty-one, and through the first ring in nineteen. In fourteen cases the opening was through the second ring, and in only two below it. In cases of laryngeal carcinoma in which the growth can be removed through laryngofissure, the level of the tracheal opening is not of great concern, so long as the opening is sufficiently far removed from the wound in the larynx to avoid adding to the irritation; the tube, as a rule, can be dispensed with after a few days. When palliation only is anticipated in cases of laryngeal malignancy the trachea should be opened as low as possible. The same is true in all other cases in which the tube must remain in the trachea for a long period. Laryngeal stenosis may result from high tracheotomy, or if stenosis is present it may be aggravated by the resultant irritation and infection, together with the accompanying granulations.

The size of the tracheal cannula obviously will depend on the size of the tracheal lumen in the individual case. Generally speaking, it is preferable to use a reasonably large tube, since the mu-

coid secretion can be more readily expelled and there is less likeli-
hood of emphysema developing, providing the tube fits the tracheal
window snugly. The possibility of trauma to the tracheal mucosa
is no greater with the large than with the small tube, nor is the
difficulty of decannulating increased. If tracheotomy is done pre-
liminary to laryngectomy, I prefer to use a comparatively small
tube, in order to conserve the cricoid cartilage or the cartilage of
the tracheal rings. The common tendency with most surgeons of
limited experience is to use too small a tube. The length of the
tracheal cannula is a greater factor in producing irritation of the
trachea than is its caliber. With the newer type of adjustable tra-
cheotomy tube the length can be satisfactorily accommodated to
the patient and reduced or increased as is necessitated by the
edema about the wound.

Comparatively few conditions were encountered during trache-
otomy in this group of cases that complicated the procedure appre-
ciably. The most serious of these, cessation of respiration, oc-
curred in five cases. In one of these cases, that of a child one
year of age, with a foreign body in a bronchus, fatal broncho-
pneumonia subsequently developed. The remaining four patients
recovered. One of them, a child 2½ years of age, stopped breath-
ing during direct laryngoscopic examination made to determine
the cause of dyspnea. An opening was immediately made through
the thyroid cartilage, and after respiration started the opening
was lowered. A malignant thyroid gland overlay the upper por-
tion of the trachea and was attached to it in each of two instances,
necessitating a transverse incision and the coring out of a portion
of the malignant mass in order to expose the trachea. An abscess
of the anterior part of the neck was encountered in two instances
and the trachea was opened through this; both patients recov-
ered. The trachea was found markedly displaced by a carcinoma
of the thyroid gland in two cases and was difficult to find. In two
instances such marked calcification of the tracheal cartilages was
present that it was impossible to cut them with a scalpel. Both
patients were men, respectively 57 and 58 years of age. Suf-
ficiently active bleeding to be of serious consequence was not
encountered in any of the cases at the time of operation, even in
those in which tracheotomy was performed without anesthesia,

although troublesome oozing occurred in a few instances. Neither was serious secondary bleeding encountered. In this connection the report of Schlaepfer is interesting, since he pointed out a danger associated with tracheotomy that is commonly overlooked. He reported two cases of fatal secondary hemorrhage respectively on the seventeenth and ninth days following tracheotomy. Both of these resulted from delayed perforation of the innominate artery.

Providing dyspnea of cardiac or pulmonary origin has not been mistaken for the obstructive type, and providing tracheotomy has not been too long delayed, complete relief from the respiratory difficulty should follow the operation. The immediate results of few surgical procedures are more gratifying than those following definitely indicated tracheotomy. Frequently a child, nearly exhausted from labored respiration, and almost moribund from anoxemia, will drop off into restful sleep within a few minutes following tracheotomy, even though the operation is carried out without anesthesia. Failure to secure complete relief from dyspnea on opening the trachea and aspirating the accumulated mucopurulent secretion is always of ominous portent, since it means either that the diagnosis has been erroneous or that tracheotomy has been too long delayed and cardiac failure or bronchopneumonia has supervened. In one of the cases in the series under consideration the dyspnea resulting from hemorrhage into a wound of recent thyroidectomy was not relieved on opening the trachea. The patient died of cardiac failure shortly after tracheotomy. In the case of myasthenia gravis previously mentioned the obstructive symptoms completely disappeared after the trachea was opened, but because of weakness of the respiratory muscles the patient was unable to carry on breathing for twenty-two days without the aid of the Drinker respirator. Another patient with bilateral abductor paralysis secondary to carcinoma of the esophagus continued to have difficult breathing after the trachea was opened, until an extra long tracheotomy tube was inserted. The trachea was displaced to the right in this case, and the tube encountered marked resistance about 5 cm. below the tracheal opening. After the tube had been passed beyond this point, complete relief was secured.

In two instances, foreign bodies were removed through the tracheal opening following tracheotomy. In another case the trachea was opened for the purpose of removing a safety pin, but this was coughed into the nasopharynx while an attempt was being made to grasp it. It was readily removed from the latter situation.

Tracheotomy done reasonably early, in a hospital that is well equipped for treatment of diseases of the nose and throat, involves comparatively little risk. The hazard is greatly increased by delaying the procedure, especially in cases of goiter. It is as essential, however, that a nursing staff and interns accustomed to cases of this type be in charge, as that the operation itself be done early and properly. During the first few days it is absolutely necessary that the patient be watched carefully because of the possibility of the tube slipping out of the trachea or becoming obstructed with tenacious mucus, and that these conditions be recognized immediately. Death may result otherwise.

Although the complication which most frequently follows tracheotomy is bronchopneumonia, this condition occurs less often than is generally supposed. It is more likely to develop if patients are debilitated, especially if they are of advanced years, and if they have had urgent dyspnea for a prolonged period. Bronchopneumonia was encountered in only thirteen cases (6.3 per cent) in this series. Six of these patients were more than 50 years of age, and three of them were aged more than 65 years. In two cases the pneumonic process was thought to have started prior to opening of the trachea, and in two others it probably was a terminal affair. The heart of one patient was fibrillating before removal of a substernal exophthalmic goiter and tracheotomy that was necessary a few hours later. The patient died of bronchopneumonia twenty-four hours subsequently. A child, 1 year of age, was brought to the hospital in extremis, with a history of sudden onset of severe dyspnea two days previously. Bronchoscopic examination had been done elsewhere. The patient almost died before the trachea could be opened, and some cyanosis continued after tracheotomy. Death from bronchopneumonia occurred three days later. The prognosis in cases of extensive bronchopneumonia following tracheotomy is always grave. Only three

patients in this group of thirteen recovered. Mediastinitis was not encountered in a single instance in the series, although the mediastinum was apparently opened into at the time of operation in one case in which, because of a neoplasm, it was necessary to open the trachea very low.

A man, 72 years of age, who had an epithelioma of one vocal cord, and a history of coronary disease, had repeated attacks of angina pectoris during the course of a number of weeks after the trachea was opened. Continuance of the attacks, and the marked prostration incident to them, finally made it advisable to send the patient home without removing the laryngeal lesion. On his return to the clinic about a year later the growth was removed and the tracheal opening closed without incident.

There was a total of eighteen deaths (8.7 per cent) in the series of 206 tracheotomies. Fifty-five per cent of these resulted from bronchopneumonia; 11 per cent were the result of cardiac disease. Among the 133 cases of carcinoma of the larynx there were three deaths (2.2 per cent) following tracheotomy. One of the three patients was a man, 69 years of age, who came to the clinic with severe obstruction from an extensive, inoperable growth. Another death resulted from streptococcic septicemia which followed tracheotomy done four days after the first stage of laryngectomy. Among the thirteen cases in which foreign bodies were present, and tracheotomy was required, there were two deaths, a mortality rate of 15.3 per cent. Two of the four patients with acute laryngotracheitis died.

The length of time that maintenance of the tracheal opening was necessary varied greatly in different cases. The shortest period was two days in a case in which thyroidectomy was performed. Laryngeal examination in this case immediately after operation disclosed that the vocal cords were moving normally. Twenty-three hours later they were both fixed in the median line and tracheotomy was necessary to relieve the marked respiratory distress. Two days later the cords were again functioning normally and the tracheal cannula was removed. Two other patients whose tracheas were opened following bronchoscopy and after thyroidectomy wore their tubes for only three days. As a matter of fact, the only interest attached to these cases centers about the

prompt restoration of function of the vocal cords in one instance, and the rapid clearing of edema within the larynx in the other two. A difference of a few days, or of even a week or two, in the time of removal of a tracheal cannula is of little consequence as far as the patient's general health is concerned. Patients who have undergone laryngectomy and who carry on respiration through the open trachea for years rarely have trouble in this regard. Woods and Thomson reported the case of a woman, 81 years of age, who had worn a tracheal tube for more than seventy years. It was noted that the patient never had suffered from bronchitis. The proportion of cases in which tracheotomy is permanent, compared with those in which it is only temporary, will vary greatly in different series, depending on the relative number of malignant and inflammatory lesions included. In the group of cases under consideration the tracheal opening was maintained permanently in 114 cases and temporarily in sixty-nine. In six cases the tube is still being worn. Although from fourteen months to four years and five months have elapsed since its introduction, in most of these, eventual removal of the cannula is anticipated. All of the conditions necessitating opening of the trachea in this group were benign, one of them being collapse of the trachea following thyroidectomy, and five multiple papillomas; three of the patients have been well for more than six months. Neither these six patients who are still wearing cannulas, nor those who died, are included in the present consideration. The decided preponderance of permanent tracheotomy as compared with temporary tracheotomy in this series is because of the large number of cases of laryngeal carcinoma. In addition to cases of the sort just mentioned, permanent tracheotomy also was required in three cases of bilateral paralysis of the vocal cords after thyroidectomy in which operation had been performed in from one year to four years and eight months before examination in the clinic and in two cases of amyloid tumor of the larynx. One of the latter patients wore his tracheal tube until he died three years later from an entirely unrelated condition. The laryngeal lumen of the other patient with an amyloid tumor has greatly increased in size under treatment, and the patient may eventually be able to carry on respiration in the normal manner.

One girl, 7 years of age, whose trachea was opened three years ago because of obstruction caused by multiple papilloma, has been free from growths for more than two years, and the tube has been corked for twenty-seven months. Although this patient's cannula might be removed at any time, it has been deemed safer to postpone removing it for a time longer, since breathing space is slightly restricted and might give some trouble should an acute inflammatory process develop.

In most instances decannulation was preceded by partially and then completely closing the tube with a rubber stopper for a few days as suggested by Jackson. This is particularly satisfactory in children, although in a few cases a smaller tracheal tube was inserted for a few days and then this was corked before removal of the tube. Even though the tube is worn for several months, the fistulous tract usually heals promptly. Excision of the epithelized tract, with surgical closure, was carried out in nine cases. The longest period that the tracheal tube was worn without surgical closure becoming necessary later was four months; one patient went seventy-two days, and two others fifty-two and fifty-four days respectively. The shortest period of cannulization, with necessity of subsequent surgical closure, was four months. Considerable scarring of the neck was present in these cases. In another case in which surgical closure was required the tracheal tube had been worn for only a month following laryngofissure and diathermy for a carcinoma of the larynx of a patient 68 years of age. Convalescence in this case was prolonged because severe nephritis supervened, and a slough occurred at the site of the wound in the thyroid cartilage with subsequent formation of a fistulous tract at this point rather than at the tracheal opening.

SUMMARY.

A review of 206 tracheotomies performed on 200 patients is presented. Seventy-one of these were emergency procedures; 135 were elective. The most frequently encountered primary pathologic conditions necessitating tracheotomy were: carcinoma of the larynx (133 cases), goiter (twenty-five cases), foreign bodies in the tracheobronchial tree (fourteen cases), and multiple papil-

loma of the larynx (eight cases). Although there was no imme-
diate surgical mortality directly ascribable to the operation,
eighteen patients died subsequently. Bronchopneumonia was the
most frequent postoperative complication. It occurred in only thir-
teen cases in the entire series, and probably was present in two of
these before opening of the trachea. Only three patients among the
thirteen recovered. Mediastinitis was not encountered in any case.
The deaths in the series were almost entirely the result of dyspnea
and delay in opening the trachea rather than of the tracheotomy
itself.

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BIBLIOGRAPHY.

1. Arbuckle, M. F.: Cicatricial Stenosis of the Larynx. Arch. Oto-
laryngol., 8:686-697, (Dec.) 1928.

2. Jackson, Chevalier: Treatment of Laryngeal Stenosis by Corking the
Tracheotomic Cannula. Laryngoscope, 29:1-4, (Jan.) 1919.

3. Schlaepfer, Karl: Total Hemorrhage Following Tracheotomy for
Laryngeal Diphtheria. Jour. Am. Med. Assn., 82:1581-1583, (May 17)
1924.

4. Thomson, St. Clair: Tranquil Tracheotomy, by Injecting Cocaine
Within the Windpipe. Brit. Med. Jour., 2:460-461, (Oct. 11) 1919.

5. Thomson, St. Clair: Tracheotomy Tube Worn for Over Seventy
Years. Brit. Med. Jour., 1:778, (April), 1925.

6. Woods, Robert: Tracheotomy Tube Worn for Over Sixty Years.
Brit. Med. Jour., 1:733, (April 18) 1925.

XV.

SIGNIFICANT ANATOMIC FEATURES OF THE AUDITORY MECHANISM WITH SPECIAL REFERENCE TO THE LATE FETUS.

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II. OBSERVATIONS—(Continued).
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B. PARS PETROSA.

Gross Observations.—If the dura be stripped from the pars petrosa of the six-months fetus the independence and isolation of this element of the skull is revealed. The dura adheres tenaciously to the infero-anterior and infero-posterior borders of the bone and also to the apical region, to the internal auditory meatus and the fossula of the saccus endolymphaticus. Elsewhere it may be stripped away relatively easily. The removal of the dura exhibits wide fissures demarcating the pars petrosa from the neighboring bones of the skull. The anterior fissure separating the otic capsule from the greater wing of the sphenoid contrasts with the closer union (synchondrosis sphenopetrosa) of the adult. The posterior fissure (fissura petro-occipitalis) separates the pars petrosa from the pars basilaris of the occiptal bone. This fissure, though not so wide as the anterior fissure, is nevertheless far from the close synchondrosis of the adult.

The long axis of the pars petrosa of the six-months fetus, as in the adult, runs diagonally in the floor of the skull anterome-

dially. Measured along the superior angle, the pars petrosa is be-
tween 17 and 18 mm., the length varying in different specimens
of supposedly the same age.

The pyramid presents two faces within the skull, namely, an-
terior and posterior, as in the adult. The superior angle, sepa-
rating the anterior and posterior surfaces, does not lie in perfect
alignment with the course of the superior petrosal sinus and is a
little anterior to the attachment of the tentorium cerebelli. The
plane of the tentorium strikes the arcuate eminence of the su-
perior semicircular canal midway in its circuit. The anterior sur-
face of the pars petrosa presents a prominent rounded eminence
upon its medial third, produced by the contour of the coils of the
cochlea. The lateral third of this face also presents an eminence,
but one of less size. It is formed by the contour of the double
ampullæ of the superior and horizontal semicircular canals. Be-
tween these eminences the middle third of the anterior face is de-
pressed and in the depression lies the exposed geniculate ganglion.
From it may be traced anteromedially the great superficial pe-
trosal nerve as it courses toward the apex. Other landmarks of
the anterior surface may be studied in better detail from the
model than from a freshly dissected preparation. Here occurs
the long unclosed facial canal in which in the fresh state
the fusiform geniculate ganglion lies. Posterior to this lies a
rounded bony eminence. Microscopically this presents a picture
of more mature bone than the neighboring capsule with which,
however, it fuses. This protuberance is interpreted as a vestigeal
"osselet," a remnant of the primordial skull as described by
Augier (Fig. 7, pr). More inferiorly the open groove for the
facial nerve, continuing from the fossa of the geniculate ganglion,
descends almost vertically to enter the middle ear cavity.

The flat posterior surface of the pars petrosa faces more supe-
riorly than is the case in the adult. The conspicuous opening of
the internal auditory meatus lies near the superior part of the
face. It is oval in shape and measures 2 by 3 mm., its long axis
extending from an inferomedial position to a superolateral one.
Because of its relatively high position one can readily see into
the depths of the meatus in looking down from above. When the

contained nerve fibers have been pulled from the meatus, its fundus is seen, as in the adult, to consist of a superior and an inferior area. These areas are not definitely separated as in the adult by a crest, however, but by a bulging posteriorly of the anterior wall of the meatus. A deep groove, the sulcus facialis, leads from the fundus upon the anterior surface. The superior vestibular area of the fundus lies at a relatively higher level at this age than it does in the adult. Posterior to the cochlear area a slight depression in the inferior vestibular area marks the foramen singulare. Anteromedial to the internal auditory porus, the flattened posterior face of the pyramid corresponds to the base of the cochlea. Posterolateral to the porus the posterior semicircular canal forms a low prominence in the posterior cranial fossa, the greatest convexity of its arc being contiguous with the adjacent part of the squamous bone. Corresponding to a spot just below the crus commune is a minute depression, the external aperture of the vestibular aqueduct, which marks the location of the saccus endolymphaticus. As may be seen in the microscopic sections, the saccus is unprotected by any operculum of bone at this age.

The arc of the superior semicircular canal stands out in bold relief from the superior aspect of the pyramid, reaching the anterior surface and being separated from the squama by a mass of cartilage to which the canal is adherent. The height of the canal is between 3 and 4 mm. Its transverse diameter is 1 cm. This latter measurement may also be considered as the greatest transverse measurement of the pars petrosa. The prominence of the canal is partly tunneled by the large subarcuate fossa described below.

As is well known, the interior of the pars petrosa is hollowed out to form the bony labyrinth. The labyrinth consists of the two highly specialized divisions, the cochlea and the vestibule, as in the adult. The measured distance of the hollow cochlea from base to apex in the six-months fetus, specimen No. 3887, is 5 mm. The diameter of the base of the cochlea is 7 mm. These measurements are identical with those made upon an adult. The socalled height of the apical coil, as seen in Section 330, Fig. 16, is 2.25 mm. It must be remembered that the cochlea lies in the skull with its modiolar axis in a nearly horizontal plane, the apex

inclining slightly downward. At this age, however, the apex is relatively lower than is true for older specimens.

A cone-shaped network of bony trabeculæ supports the spiral ganglion and forms the modiolus. A delicate double-layered bony plate projects from the modiolus into the middle of each coil, forming the osseous spiral lamina. Right-angled trabeculæ connecting these plates are not yet well formed. It was found impossible to measure exactly the height of this structure in this fetus. From the study of the serial sections, however, one is led to conclude that the modiolus has not yet reached its full height. The height of the modiolus in a two-months child was 3.2 mm. The base of the modiolus is much perforated for the entrance of the cochlear nerve.

Just within the round window the basal turn exhibits a funnel-shaped depression which is the entrance to the cochlear canaliculus. The diameter of the lumen is 0.25 mm. In this specimen the canal could not be followed all the way to an external aperture. Anterior to the cochlear canal is an equally large bony-walled canal carrying a vein, probably the v. canaliculi cochleæ. This canal extended through to the exterior of the pars petrosa.

The vestibular division of the bony labyrinth consists of a smooth-walled irregularly elongated cavity into which open the three semicircular canals, which may be considered as diverticuli of it. The vestibule communicates with the cochlea where the scala vestibuli curves down into it and becomes continuous with the cisterna perilymphatica. This relatively wide communication (height 2 mm.) can readily be studied in the model by piling Blocks, 1, 2 and 3. The vestibule communicates with the surface of the otic capsule by way of the aqueductus vestibuli whose lumen measures 0.677 by 0.058 mm., as seen in Section 203, where the aqueduct is following along the crus commune. This measurement is not that of the lumen of the endolymphatic duct, which is much smaller (see below).

The vestibule, measured from the pit of the posterior semicircular canal ampulla to the wall of the saccular recess, as seen in Fig. 16, Section 330, is 6 mm. The longest diagonal distance in this section from the pit of the posterior ampulla to the outer

wall of the superior canal is 8.5 mm. The anteroposterior width of the vestibule at this level is 5 mm. The supero-inferior dimension is 2.8 mm. When the labyrinth is oriented as in the adult this latter dimension will be the mediolateral dimension, and the first dimension given, 6 mm., will represent the height. The diameter of the superior semicircular canal measures on its lateral limb 0.935 by 1.29 mm. and on its medial limb at the level of the sacculus 1.61 by 1.64 mm. (Section 153). The ampulla of this canal measures 1.64 mm. in an anteroposterior direction (Section 330) and 2.3 mm. in a supero-inferior direction. Its demarcation from the utricle is not obvious in the sections, therefore a lateromedial measurement cannot be given. As may be seen in the microscopic sections, the lateral limb of this canal is abnormally flattened.

The ampulla of the lateral semicircular canal measures 2 mm. supero-inferiorly by 2.5 mm. anteroposteriorly by 2.03 mm. lateromedially. This is quite the largest ampulla. The lumen of the peripheral part of the lateral canal measures 1.4 by 1.6 mm. The nonampullated end is malformed.

The ampulla of the posterior semicircular canal measures 1.9 by 1.2 by 2.2 mm. The measurement of the peripheral section of the canal is 0.74 by 2.03 mm.

Microscopic Observations.—The otic capsule is at this time encrusted by a thin shell of bone which flakes off only too readily with a sharp scalpel. Under the dissecting microscope, this layer of bone presents a delicately mottled pattern. Beneath this shell-like layer one encounters a granular substance, the marrow-containing bone described below. Through this can be seen the outline of the membranous labyrinth in places, for the bony capsule is still translucent. The membranous superior canal is particularly obvious.

In the sixth month of fetal life, the three types of bone described by Bast, Eckert-Mobius, et al., which comprise the inner capsule, are markedly differentiated. The periosteal layer, endosteal, and enchondral (intrachondral, Bast) are all easily distinguished. The periosteal layer exhibits a lattice-like structure. Its long, more or less parallel, trabeculæ are interspersed with spaces

containing delicate areolar tissues with occasional minute capillaries. True marrow does not occur here yet, as in the interosseous spaces of the enchondral bone. The trabeculæ are dotted with the blue-staining (H. and E.) bone cells which vary in shape much as do any fibroblasts. Some appear triangular, some oval, and others rounded. Each cell is surrounded by a clear space, the lacuna, across which strands of protoplasm extend into the canaliculi. Almost no empty lacunæ are to be seen, indicating that the bone is in a living state. The trabeculæ are bordered by osteoblasts (Fig. 20). These cells are of about the size of the bone cells. They are blue staining throughout and carry relatively little cytoplasm. For the most part these cells are aligned with their long axes parallel to the trabeculæ. Occasionally they are at right angles. Here and there large multinuclear cells with pink staining cytoplasm and blue standing nuclei are encountered. They are identical in appearance with those seen in the tympanic sulcus. These are osteoclasts (Fig. 21). The periosteal bone is the layer which flaked off so easily in the gross dissection. Evidently periosteal bone cells, once started to develop, mature rapidly. It is well known that the membrane bones appear relatively late in the history of the development of the skull. This "fiber bone" in the microscopic sections seems more like mature bone than either of the other two layers.

The endosteal layer appears to be next to the periosteal layer in degree of maturity. The two layers comprising it are readily distinguished in specimen No. 3887. The inner layer takes a bluish stain with hematoxylin and eosin, the outer, a pink. The marked contrast between endosteal and periosteal bone is readily seen in Section 330 (Fig. 16), around the superior semicircular canal. Here no third type of bone intervenes between the two. The inner layer of endosteal bone is lined by approximately five layers of endosteal cells whose nuceli are minute, being one-third to one-half the size of the nuclei of the osteoblasts occurring elsewhere. The endosteal cells form strands which jut into the perilymphatic spaces and make up its reticular structure. Occasional giant cells occur as in the periosteal bone. The wall of the lateral limb of the superior canal is in one place covered only by endosteal bone (Section 185, Fig. 19). As the model clearly shows (Figs. 5 and

6), this canal is abnormally flattened, giving it an angular contour. The median limb of the horizontal canal is also irregular in contour. Apparently some disturbance of the endosteal bone has caused this anomaly.

The third type of bone is the enchondral. This is composed of tortuous trabeculæ which have a light staining matrix, tinged with blue (H. and E.). In this lie the single, double or even multinucleate cells, such as Bast describes. These appear to have formed around themselves accretions of calcium. By focusing with high power through a well-cleared area one gets the impression that these rough-hewn ovoid bodies protrude into a canal-like region. Sometimes these accretions bridge across to the opposite side. Usually, however, they appear as a series of crude rounded protuberances bordering on the interior walls of the other layers. They form a series of tortuous trabeculæ between the other two layers of bone rather than "cartilage islands." The trabeculæ are bordered by osteoblasts. These cells are aligned with the long axis at right angles to that of the trabeculæ. Occasional osteoclasts occur. The trabeculæ are surrounded by numerous marrow spaces. In the six-months fetus the marrow is definitely hematopoietic in this region. This type of marrow shows as the irregular black splotches in Fig. 16. In older specimens there are certainly fewer red blood cells in the marrow of this area than in the depths of the mastoid, for example.

Persistence of Cartilaginous Areas.—In the six-months premature infant it is practically impossible to study the centers of ossification as the majority of these have coalesced at this time. Much more striking to the observer are the unossified centers of cartilage (Figs. 16 and 19). The mass of cartilage illustrated is somewhat spherical. It lies under the arch of the superior canal and between it and the posterior canal. It measures approximately 4.5 mm. in diameter. Extensions taper above and below the main mass. One specimen, aged 1 year, showed a small mass of cartilage under the superior canal. The persistence of cartilage in this area seems to occur in those cases where a large subarcuate fossa persists.

A second mass of cartilage is present in the fissura antefenestram. This may be seen in the model (Fig. 11). The carti-

lage borders on the internal vestibular wall, a point not made clear
in the model. A fossula post-fenestram was not located in speci-
men No. 3887. The whole footplate of the stapes is covered with
cartilage at this age. The capitulum of this ossicle is composed
of cartilage. The handle of the malleus also is cartilaginous. The
whole length of the auditory tube is of cartilage at this stage. In
specimens ranging in age from birth to 2 months an elliptical
mass of cartilage was seen in the hypotympanum. This was
interpreted to be the proximal end of the styloid process. These
masses of cartilage have all disappeared, usually by the end of the
first year.

Fossa Subarcuata.—The subarcuate fossa of the fetal and in-
fant temporal bone is a very obvious feature (Fig. 19). As the
name implies, it is a cavity under the arch of the superior semi-
circular canal. The entrance to the fossa in this specimen has a
diameter of 2 mm. The depth of the fossa is 5 mm. It is filled
with dura, which has a good vascular supply. In adult man this
cavity is generally obliterated, although occasionally it remains.
In man, when present, it is entirely filled with dura, through
which course a vein and an artery.

Thirty-five cases were studied in order to obtain an idea of the
extent of the fossa and its time of obliteration. The results are
listed in Table II.

The table shows that the fossa usually becomes much reduced
in size by the end of the first year. Occasionally, however, the
depression may be seen on an adult dried skull. The study further
revealed that the direction and size of the fossa differs in different
individuals, and even on the two sides of the same individual. In
some specimens the cavity dips under the superior canal and comes
to the cranial surface again on the other side of the canal, the
dura being continuous throughout. In five cases the cavity was
found to communicate with the antrum, the dura actually serving
as a lining for the antrum. In four instances this occurred on the
left side only. In one it occurred on both sides.

Canalis Facialis.—When the facial nerve leaves the internal
auditory meatus, in the six-months fetus, it proceeds unprotected
in a groove on the superior aspect of the pars petrosa to the mid-
dle cranial fossa instead of into the tympanum, as in older speci-

TABLE II. CLOSURE OF SUBARCUATE FOSSA.

No.	Age	Race	Sex	Width		Height	Depth
4151	26½ hours	W	F	Right	3 mm.	3 mm.	2½ mm.
				Left		3 mm.	3.9 mm.
3324	Stillborn 4 da. prem.	N	M	Right	2.2 mm.	2.6 mm.	5 mm.
				Left	2.2 mm.	2.2 mm.	5 mm.
4331	1 day	N	M	Right	1 mm.	2.8 mm.	7 mm.
				Left	3 mm.	3 mm.	6 mm.
3337	10 days	W	F	Left	3 mm.	3 mm.	6 mm.
4139	15 days	W	M	Right	5 mm.	3 mm.	6.5 mm.
				Left	3 mm.	3 mm.	7 mm.
4330	19 days	W	M	Right	2.7 mm.	3.2 mm.	4 mm.
				Left	3 mm.	2.6 mm.	6 mm.
4396	21 days	W	M	Right	3 mm.	.6 mm.	2.8 mm.
				Left	3 mm.	2.5 mm.	4 mm.
3849	1 month	N	M	Right	3 mm.	Series incomplete	6 mm.
3967	2 months	W	M	Right	3 mm.	?	5.4 mm.
				Left	3 mm.	2.8 mm.	6 mm.
4427	2½ months	W	F	Right		1 mm.	6.8 mm.
3849	2½ months	W	F	Right	.4 mm.	1.0 mm.	.9 mm., series incomplete
				Left		1.2 mm.	5.2 mm.
3873	3½ months	W	F	Right	2 mm.	?	6 mm.
3865	4 months	W	M	Right	1.8 mm.	Series incomplete	6.5 mm.
				Left	1.2 mm.	2.4 mm.	6 mm.

TABLE II. CLOSURE OF SUBARCUATE FOSSA.—(Continued)

No.	Age	Race	Sex	Width		Height	Depth
4078	5 months	W	M	Right	3 mm.	3.4 mm.	11 mm.
				Left	4 mm.		6 mm.
3920	5½ months	W	F	Right		3 mm.	6.1 mm.
				Left		3 mm.	6.2 mm.
3947	7 months	N	F	Right	1 mm.	3 mm.	7.8 mm. Emerges to surface;
				Left		2.6 mm.	meets antrum.
3923	10½ months	N	M	Right	1 mm.		2.0 mm.
				Left	closed		
4106	1 year	N	F	Right	1 mm.	.22 mm.	5.0 mm.
				Left	1 mm.	2.5 mm.	7.5 mm. 0.5 between antrum and dura.
4338	1 year	W	F	Right	1 mm.	2.8 mm.	6.5 mm.
				Left	1.7 mm.	4 mm.	5.5 mm.
4048	13 months	W	M	Left	1 mm.	2.5 mm.	9 mm.
II.	13 months	W	F	Right	2?	2.2 mm.	5 mm. Communicates with
				Left	2?	2.5 mm.	3.4 mm. antrum.
3878	1 yr., 4 mos.	W	F	Right	1 mm.	4 mm.	6 mm.
4278	1 yr., 8 mos.	N	F	Right	6 mm.	1.4 mm.	11 mm. Also communicates with dura.
				Left	7 mm.	2.8 mm.	13 mm. Extending posteriorly and superiorly to cranial region.
4457	2 years	W	F	Left	closed		

					8 mm.	8 mm. and with antrum.
3951	5 years	W	F	Right, just closing Left 1 mm.	8 mm.	8 mm. and with antrum.
3903	8 years	W	M	Right, area not seen Left closed		
3956	10 years	W	F	Right closed Left persists but could not be measured.		
4336	12 years	W	M	Right closed Left still persists (narrow). Leads into mastoid—meningitis.		
3948	13 years	N	M	Closed		
4056	31 years	N	M	Closed		
3884	35 years	W	M	Right, small groove present Left, small groove present		
4486	40 years	N	F	Left, slight groove present		
3972	44 years	W	M	Right closed Left closed		
3897	45 years	W	F	Left .4 mm.	.2 mm.	1 mm.
3917	47 years	N	F	Left closed		

The item "width" may be taken to represent the diameter of the fossa as the opening is practically circular.

mens. This might be anticipated, however, if one recalls the relative positions of otic capsule and cranial fossæ. During the proximal upper part of its course in the cranial fossa the nerve bears the geniculate ganglion. In fact, the ganglion cells begin to appear before the nerve completely leaves the meatus (Section 264). The cell bodies continue to appear until Section 331—i. e., a distance of 1.3 mm. Their presence gives the nerve a fusiform structure at this level. The ganglion cells are divided by sheaths into six or seven groups (324-329). One group, consisting of very few cells, lies in the posterior part of the nerve fibers. All the other groups lie in the anterolateral region of the nerve. A few ganglion cells trail in horizontal plane along the course of the superficial petrosal nerve for a short distance. This nerve is likewise uncovered by bone.

Not until Section 374 does the facial nerve become completely surrounded by bone. At this point it is running parallel to the stapedial footplate and is just entering the tympanum. At about this level (Section 368) may readily be seen some of the periosteal bone from the squamosa entering into the formation of a part of the tegmen and in close relation to the facial nerve. The nerve is enclosed for a distance of 7.6 mm. It enters the tympanum while running parallel to the stapedial footplate. As it courses posteriorly across the tympanum it again lies in an open groove as described under "Tympanum."

For comparison and verification of these observations the anatomic status of the facial nerve was then studied in younger and in older specimens, as well as in certain animal material. Specimen No. X (age less than 2 months' embryonic life) shows the facial nerve coursing along the otic capsule. Not even a shallow groove is seen on the capsule, marking its course, and certainly no indication of enclosure of the nerve is present at this age. Specimen No. XV, M256 (2¾ months fetus) gives the same picture. Specimen No. XXI (3-months fetus) shows a shallow depression in the capsule under the course of the nerve. Specimen XVI (4-months fetus) shows the nerve surrounded by very dense connective tissue. The capsule is hollowed slightly in a tiny groove, where the nerve starts its descending course. Specimen XVI (7-months fetus) is even less protected than in No. 3887.

Table III presents a study made from forty older cases. When possible, both right and left bones were examined. Thus a total of sixty-three temporal bones were examined.

The table indicates that apertures are constant and relatively large in early infancy and that after 13 months of age they occur with less frequency and are relatively smaller. With maturity they usually disappear, except for a minute opening for a blood vessel. Occasionally a sizable aperture persists in the adult. The location of the aperture occurs most frequently where the facial nerve crosses the oval window and descends, posterior to the niche of the window. The bar of bone enclosing it grows around the nerve much as the bony promontory envelops the round window. Occasionally an aperture may open, not into the middle ear proper but into an accessory middle ear cell.

Interesting observations in regard to this point were made upon certain animal material. See Discussion.

C. MEMBRANOUS LABYRINTH.

The membranous labyrinth of the six-months, premature infant is well developed, as may be seen in Fig. 16, Section 330. Fig. 1, Section 304, presents the labyrinth in the negative and Figs. 8, 9, etc., depict is as it appears in the model.

It was primarily with the idea of studying the relationships of the endolymphatic system to the perilymphatic system that the model was constructed in the particular manner illustrated. Models have been cast of the endolymphatic system alone and of the perilymphatic system alone in previous investigations by various students of the subject. Here the attempt has been made to show in one model both the endolymphatic and perilymphatic systems by the simple expediency of cutting out from the wax plates the perilymphatic system. This leaves the endolymphatic system in solid wax, as is also the bony capsule. Since the ductus endolymphaticus could not be traced in the bony capsule without cutting it out, it was decided to represent the saccus as a hollow cavity lying in solid dura (Figs. 3 and 4).

Fig. 16 clearly shows the membranous labyrinth, consisting of an endolymphatic and a perilymphatic system, each system occurring in both the cochlear and vestibular divisions of the labyrinth.

TABLE III. INCIDENCE AND EXTENT OF APERTURES IN THE FACIAL CANAL.*

No.	Age	Race	Sex	Right Ear	Left Ear
4151	26½ hours, 8 fetal months	W	F	Vert. .9 mm. Horiz. 2.6 mm.	Vert. 1 mm. Horiz. 2.8 mm.
3824	Stillborn, 4 days premature	N	M	Vert. 1.8 mm. Horiz. 1.5 mm.	Vert. .3 mm. Horiz. .9 mm.
4331	1 day	N	M	Very minute, merely for the passage of a b. v.	
3337	10 days	W	F	Not seen	Vert. 1.8 mm. Horiz. 1.5 mm.
4439	15 days	W	M	Horiz. .6 mm.	Vert. 1.5 mm. Horiz. 2.3 mm.
4330	19 days	W	M	No aperture seen	Very small for venule
4306	21 days	W	M	Vert. 1.4 mm. Horiz. .2 mm.	Vert. 1.4 mm. Horiz. .3 mm.
3349	1 month	N	M	Very slight	
3367	2 months	W	M	Very minute for passage of small blood vessel	
4427	2½ months	W	F	Vert. .8 mm. Horiz.	Not cut
3949	2½ months	W	F	Not through region	Vert. .7 mm. Horiz. .6 mm.
3873	3½ months	W	F	Vert. .7 mm. Numerous other small apertures occur Horiz. 1 mm.	Not received

				No aperture on either side except for passage of b. v.	
3865	4 months	W	M		Minute, located posteriorly
4078	5 months	W	M	Vert. .2 mm. Horiz. .9 mm.	Vert. .5 mm. Horiz. 1 mm.
3920	5½ months	W	F	Vert. .2 mm. Horiz. 1 mm.	Vert. .6 mm. Horiz. .4 mm.
3947	7 months	N	F	Very slight. Located posteriorly in descending course accompanying stapedius m.	None found
3923	10½ months	N	F	None found	
4106	1 year	N	F	Two small apertures on each side, one into niche of oval window, one more posteriorly.	
4338	1 year	W	F	Vert. .2 mm. Horiz. .4 mm.	Vert. .2 mm. Horiz. .5 mm.
4048	13 months	W	M	Not received	Vert. .12 mm. Horiz. .5 mm. Very minute aperture post.
II.	13 months	W	F	Very minute	None seen
3878	1 year, 4 months	W	F	Vert. .4 mm. Horiz. .1 mm.	Not sectioned
4278	1 year, 8 months	N	F	Vert. .4 mm. Horiz. .1 mm.	Present but impossible to measure because of tear.
4457	2 years	W	F	Not cut	None seen
3951	5 years	W	F	Present but very small	.2 mm.
3903	8 years	W	M	1 mm. by 1 mm.	1 mm. by 8 mm. along horiz. canal inferior border.

TABLE III. INCIDENCE AND EXTENT OF APERTURES IN THE FACIAL CANAL.—(Continued)

No.	Age	Race	Sex	Right Ear	Left Ear
3956	10 years	W	F	Two small apertures	None seen
4336	12 years	W	M	Two minute ones for passage of venule and arteriole	None seen
3948	13 years	N	M	None seen, very large facial canal	Vert. .1 mm. Horiz. .6 mm.
4056	31 years	N	M	1 mm by .12 mm.	Present but could not be measured.
3884	35 years	W	M	None found	
4486	40 years	N	F	Not cut	Very thin wall. Slight aperture for b. v.
3972	44 years	W	M	None seen	Several very minute apertures.
3897	45 years	W	F	Not sectioned	None seen
3917	47 years	N	F	Not sectioned	.6 mm. by .5 mm.
4098	48 years	N	F	None seen	Not sectioned
3894	51 years	W	M	Not through area	Small aperture posteriorly
4152	69 years	N	F	Not cut	None seen
4164	73 years	W	M	Not cut	None seen

*Vert. refers to verticle; Horiz., horizontal; b. v., blood vessel; ap., aperture; post., posterior; and m., muscle.

Endolymphatic System.—The cochlear division of the endo-lymphatic system is represented by the four triangular spaces seen in the cochlea of Fig. 16, and a fifth irregularly shaped space seen at the apex. The continuous course of these triangular segments comprises the ductus cochlearis. Because of the incomplete rotation of the otic capsule we do not have at this level a cut through the mid-modiolar axis, part of which is shown, however, in Fig. 8. The endolymphatic system of the cochlea, ductus cochlearis, is confined by Reissner's membrane, membrana basilaris, and a part of the spiral ligament. The vestibular membrane (Reissner's), as seen in section extends diagonally from the axial

Fig. 16. Sec. 330. Membranous labyrinth: *cart*, cartilage; *ru,* recessus utriculi.
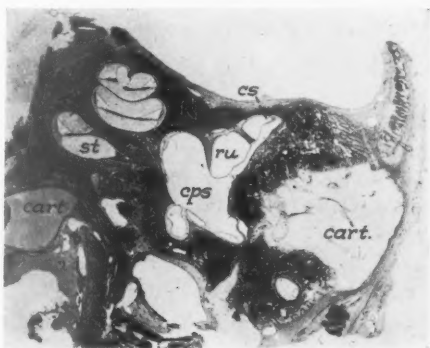
border of the limbus to the spiral ligament. The angle of its slope differs in the different coils of the cochlea, and the expanse of membrane varies accordingly. It is apparently greatest around the middle coil. Measurements made upon Section 330 (Fig. 16) show that the length of Reissner's membrane, as here seen in section, is 1.25 mm. in the basal turn. On the middle turn the more anterior division is 1.4 mm.; the more posterior division is 1.3 mm. On the superior coil the posterior division measures only 0.74 mm. The anterior division is so irregular as to make measurement impractical. Measurements made upon the lower part of the basal turn (Section 394) show Reissner's membrane on the anterior coil to be 1.2 mm. The posterior segment, where

the cochlea is curving down to the vestibule and round window
is 0.612 mm. It is thus seen that the extent and slant of Reiss-
ner's membrane is greatest in the middle of its course. Measure-
ments made upon children—2 months, 3½ months, and 8 years
old—all showed a greater length of Reissner's membrane in the
middle coil than in the upper and lower coils.

In the six-months fetus the vestibular membrane appears to
consist microscopically of a double layer of similar cells. Occa-
sionally delicate strands run from one layer to the other. The
double nature of this membrane is seen on the middle coil (pos-
terior division) in Section 325, where a line of cleavage occurs
between the two layers. Although this cleavage is an artefact
of technic, it is nevertheless informing. It has been observed by
the author in older specimens. The nuclei of the component cells
in Reissner's membrane are round and contain much granular
chromatin. The cell diameters are about .004 mm. They bulge
beyond the confines of their cytoplasm, which extends about .001
mm. on each side of the nucleus, as seen in section of the long
axis of the cell. The somewhat alternate arrangement of the
nuclei of the two layers gives one the impression that these cells
are arranged in an imbricated manner. They are undoubtedly
squamous in shape. The Reissner's membrane of a two-months
child shows the layer next the scala vestibuli with definite spindle
cells. No blood supply is evident for this membrane. Occasional
small round cells occur, but I find it impossible to tell whether
these are merely pyknotic nuclei of the component cells of the
membrane or if they are small lymphocytes. No lymph vessels
are apparent. In older specimens occasional melanophores have
been observed on this membrane. Mallory's connective-tissue stain
dyes Reissner's membrane blue with red nuclei.

The peripheral wall of the ductus cochlearis is formed primarily
by that portion of the spiral ligament which bears the stria vascu-
laris. The extent of this lateral wall varies in the different turns
of the cochlea, being greatest in the middle turn and decreasing
rapidly in length toward the apex, more slowly toward the base.
The height of attachment of Reissner's membrane in the middle
coil of the premature infant is 0.67 mm. It is 0.645 mm. for all
postnatal specimens measured. These measurements were made

on midmodiolar sections, and the specimens ranged in age from 2 months to 51 years. The measurements of the apical and basal turns show greater variations, with a slight tendency to reduction with advancing age, probably indicating a shrinkage of the tissue. In the six-months fetus the height of the stria vascularis as seen in Fig. 8 is as follows: Superior turn, posterior division, 0.548 mm.; middle coil, anterior division, 1.032 mm.; posterior division, 0.67 mm.; basal turn, 1.5 mm. In Section 394, at the lower part of the basal turn, the height of attachment of this membrane is

TABLE IV. HEIGHT OF EXTERNAL WALL OF DUCTUS COCHLEARIS IN MM.*

No.	Age	Side	Apical Coil Up.	Apical Coil Low.	Middle Coil Upper	Middle Coil Lower	Basal Coil Upper	Basal Coil Lower
3887	Fetus 6 mos.	Rt. Lft.		.548	1.032	.67	1.5 .99	.805
4330	Infant 2 mos.	Lft.		.58	.58	.645	.519	
1	?	Lft. Rt.		.516 torn		.645 .645	.548 .645	
3873	2½ mos.	Rt.		.483	.516	.645	.580	
3903	8 yrs.	Lft.		.519	.519	.645	.548	
3948	13 yrs.	Rt.		.451	.516	.387	.4·9	
3972	43 yrs.	Lft.		.419	torn		.548	
3894	51 yrs.	Rt.		.387	.612	.645	.419	

*There is evidence of vacuolization of the stria vascularis in this specimen. This probably accounts for the great discrepancy in the middle cell.

0.99 mm. on the anterior division, 0.805 mm. on the posterior division (see table IV).

Postmortem changes have so altered the normal structure of the stria in specimen No. 3887 as not to permit of cytologic study. The rhythmic occurrence of the cross section of six blood-filled capillaries in the basal turn of this region is worthy of note, however, as also the fact that a granular precipitate in the cochlear duct apparently emanating from the cells of the stria is present.

The following cytologic observations were made upon the stria of a two-months infant and upon an 8-year-old boy. The surface of the stria presents a series of regular undulations covered by cuboidal epithelium. The vestibular membrane, as seen in mid-

modiolar cut of No. 4330 (2½ months), is as follows: Upper
apical, 0.709 mm.; lower apical, 0.774 mm.; upper middle, 0.967
mm.; lower middle, 0.903 mm.; basal, 0.83. Rezius' measure-
ments for man are 0.85 mm. apical, 0.88 mm. middle, 0.81 mm.
basal. The prominentia spiralis is the most evident part of the
stria. Within its center it bears a relatively large vein, the vas
prominens, 0.0365 by 0.023 mm. A small peripherally placed
arteriole accompanies it. Dense connective tissue strands from the
membrana basilaris run under the prominentia spiralis.* Under
the crest of each small eminence in the stria is seen the cross sec-
tion of a small vessel. These occur at remarkably regular inter-
vals.

The base of the ductus cochlearis is its most important boun-
dary. It is formed by the limbus (lamina spiralis) (crista spiralis)
resting upon the membranous spiral lamina, which latter becomes
the basilar membrane. The length of the base of the ductus coch-
learis, as seen in mid-modiolar sections, was taken from the point
of origin of Reissner's membrane at the limbus, to the point of
attachment of the membrana basilaris to the spiral ligament. These
measurements, taken on Section 330 (Fig. 16), are as follows:
Superior coil, posterior (lower) part, 0.6775 mm.; middle coil,
anterior (upper) part, 0.903 mm.; middle coil, posterior (lower)
part, 0.903 mm.; basal coil, upper (anterior) part, 0.516 mm.
This cut, it must be remembered, is not mid-modiolar. For com-
parison, five other specimens were measured through midmodiolar
sections. These uniformly showed a decrease in length of the
base of the cochlear duct in proceeding from apex to base of the
cochlea. These obervations are in accord with those of Corti,
Retzius and Wrightson et al. The discrepancy found in the 6-
months fetus is probably due to the fact that this is not a mid-
modiolar cut but a tangential one of the middle coil.

The organ of Corti appears as a mound of cells arising from
the basilar membrane. Its height is greatest in the apical coil.
Measurements taken on specimen 4330 were as follows: Apical

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*The cells of Claudius with their markedly clear cytoplasm gradually
change as they mount the external sulcus, and cover the prominentia
spiralis. From this point on the cells are less perfectly cuboidal and
possess a very granular cytoplasm.

coil, lower part, 0.056 mm.; middle coil, upper part, 0.050 mm.; middle coil, lower part, 0.050 mm. Wrightson stated that the height of the plateau of the organ of Corti is 58 micra. The most prominent feature of the organ of Corti is the triangular shaped tunnel, formed by the juxtaposition of the two pillar cells. The inner pillar cell assumes a more vertical position than the outer. Within the tunnel, at the base of each pillar, is a large round nucleus and a small amount of cytoplasm in triangular shape. These are the basal cells of the pillars. The head of the outer pillar is blunt and rounded, its convex surface facing the modiolus. This convex surface fits neatly into the concave surface of the head of the inner pillar. Across the tunnel space a strand or two of nerve fibrils are occasionally seen. Just external to the outer pillar head is an irregular cavity, the outer tunnel. Lateral to this lie the three outer hair cells, sloping inward as they rise to the top of the organ of Corti, through Nuel's space. Jutting above each of these is a little tuft of so-called "hairs." The hair cells are supported by the intervening Deiter's cells. Lateral to the hair cells and Nuel's space are the rounded Hensen's cells. These gradually decrease in height until they merge into the distinctly cuboidal cells of Claudius. The latter continue across the basilar membrane until they reach the spiral ligament, where they rise to form the wall of the internal sulcus and become continuous with the cells of the stria vascularis.

On the inner side of the larger tunnel space lies the inner hair cell. Medial to this is a clear space forming the internal spiral sulcus. The medial wall of this is formed by the limbus spiralis. This structure is composed of cells whose nuclei are very definitely arranged in rows. They are so aligned as to appear much like the rows of spores seen in some plants. Occasionally all of the nuclei will be seen in the upper third of the cells, leaving the lower part of the cells clear all the way across the limbus as seen in section. Quite often the cells appear goblet shaped, with the mouths opening toward the surface. Fibrous bristles, Huschke's teeth, appear along the top of the limbus. Above and externally the limbus projects into and probably forms the tectorium, which is fibrous and stains a brilliant robin's-egg blue with Mallory's connective-tissue stain. In its outer third Hensen's

node can be seen. In some sections the tectorium is attached to the hair cells; in others it is tilted backward.

No vas spirale was observed under the organ of Corti in the human material examined. No glandular elements, such as those described by Shambaugh, were observed in the external sulcus. The fibrous extension of the membrana basilaris appeared to wall off any possibility of opening ducts.

The basilar membrane in the basal termination of its course becomes contiguous with the membrana tympani secondaria. This latter membrane dips in toward the labyrinth in an almost right-angled concavity. It is of importance to note that whereas the basilar membrane increases in length from the base to the apex of the cochlea, the vestibular membrane and the stria showed their

TABLE V. LENGTH OF BASAL WALL OF DUCTUS
COCHLEARIS IN MM.

No.	Apical Coil		Middle Coil		Basal Coil	
	Upper	Lower	Upper	Lower	Upper	Lower
3887		.677	.903	.903	.516	
4330	.806	.806	.580	.516	.483	
3873	.806	.741	.645	.600	.451	
I.	.741	.645	.600	torn	.483	
3903		.645	.580	.483	.419	

greatest measurements in the lower middle coil. Tangential measurements of the basilar membrane showed an increased length in the lower middle coil. It would seem, then, that there is an expansion of the cochlear duct in the lower middle coil or approximately that region.

The ductus cochlearis descends into the vestibule from the basal turn without observable diminution in size. Thus there hangs suspended between round and oval windows the membranous caecum vestibulare. As measured in the model this blind end is 3.5 cm. from the utricle, or in reality 1.4 mm. (Fig. 11, *dc.*) It is 1.8 cm. from the utricle in the model, or in reality, 0.72 mm. It is thus seen to hang quite free in the cisterna perilymphatica. just posterior to the attachment of the basilar membrane to the

membrane of the round window the apparently "blind sac" of the cochlear duct leads off toward the saccule by the very small ductus reuniens.

The ductus reuniens is lined by simple cuboidal epithelium, the cytoplasm of whose cells appears quite granular. The lumen measures from 0.08 to 0.12 mm. in diameter. The approximate length of the duct is 2.2 mm.

The sacculus, utriculus, the three ampullæ, with their contiguous canals, the ductus endolymphaticus and the saccus endolymphaticus, comprising the endolymphatic system of the vestibule, are well developed in the 6-months fetus. The sacculus, as seen in Figs. 8 and 9, lies close to the cochlea in the recessus sphericus. The sacculus is shaped much like an old-fashioned reticule. The anteroposterior measurement, as seen in Section 304 (Fig. 1), is 1.5 mm. The extent supero-inferiorly (lateromedially in older specimens) is approximately 1.26 mm. The mediolateral extent of the macula, as seen in the same section, is 2.16 mm. This is practically the full extent of the saccule in this direction. It measures 0.88 mm. supero-inferiorly (mediolaterally in the adult). The macula is basin shaped and faces posteriorly. It consists of a row of columnar cells, between which lie sustentacular cells. Occasionally the columnar cells are clustered, apparently arising in tufts. In rhythmic periodicity rounded spaces appear in the epithelium of the macula. Cilia-like extensions of the columnar cells are from 0.05 to 0.07 mm. in length and are colored red by Mallory's connective-tissue stain. Examination of older specimens, revealing less postmortem change, showed that the peripheral cells of the macula are low cuboidal with nuclei situated in the base of the cell. The cytoplasm appears to be a homogeneous pink-staining (hematoxylin and eosin) substance. A cuticular border is present on these cells. Upon moving toward the center of the macula, the nuclei are found a little higher from the base of the cell. The cytoplasm of these cells is a little more granular, the granules taking a blue stain. Large vacuoles are seen below the nuclei. Occasional cells in this region appear to open to the surface much as do the cells described in the limbus of the cochlea. A column of pink-staining substance projects superiorly from the nucleus and seems to extend beyond the sur-

face as the hair or cilium. As the pit of the macular depression is approached, a definite differentiation of the cells is noted. Long slender cells which are sustentacular appear between globular cells. In this material it is clear that the rhythmic spaces noted in No. 3887 are really globular cells whose whole cytoplasm has been replaced by a vacuole, leaving a relatively small peripherally located nucleus. Beyond the so-called "hairs," or cilia, which may be three-fourths of the height of the sensory cells, are blue-staining granules, the otoconia.

The inferior border (in No. 3887, medial) of the sacculus leads off to the utriculus via the utriculosaccular duct. The model when stacked clearly shows the ductus reuniens, the utriculo-saccular duct and the endolymphatic duct to be in continuous alignment with each other. The greatest curvature in the course of these small ducts occurs in the utriculosaccular duct (Fig. 8). Thus there does not appear, at this age at least, to be the Y-shaped system shown in the guinea pig. In the 6-months fetus these ducts from the ductus cochlearis take an ascending course. But if their present relationship with the rest of the capsule is maintained in the adult they will assume a more horizontal course.

The utriculus, as seen in Fig. 8, is sac-shaped with a slight constriction in the middle. In the 6-months fetus its lateral extremity fits into the recessus ellipticus of the bony vestibule. In the adult these features will be superiorly placed as a result of the rotation of the otic capsule. The greatest mediolateral extent (supero-inferior) of the utriculus may be seen in Fig. 8 and measures 4.5 mm. The width at the constriction is practically 1 mm. The macula of the utriculus occurs solely in the lateral (superior) part. The supero-inferior (mediolateral in the adult) measurement of the macula utriculi is 1.58 mm., as contrasted with 0.88 of the macula sacculi. It is thus seen that the macula utriculi is almost twice as wide as the macula sacculi. The macula utriculi begins just 60 micra beyond the macula sacculi on the superior (medial) side but extends far beyond it inferiorly (laterally). The macula utriculi extends 2.09 mm. lateromedially (supero-inferiorly) in its most protracted area (Fig. 16). This can scarcely be contrasted with the similar measurement of the saccule, 2.16 mm., since the macula utriculi extends farther medialward

(inferiorly) than the macula sacculi due to the variation in shape of the utriculus. However, as is clearly seen in Fig. 16, the macula utriculi is in a parallel position with that of the sacculus and not at right angles to it. Even in this specimen the macula utriculi covers a larger area in space than that of the saccule. The cellular structure of this macula offers no features differing from those of the sacculus so far as could be observed. The depression of the basin is more shallow.

Macula Neglecta and Endolymphatic Valve.—The location of the macula neglecta is indicated by the arrow in Fig. 9. It appears as a blunt rudimentary structure on the medial floor of the utricle. A multiplication of flat epithelial cells and an area of low cuboidal cells indicate its presence. In specimen No. 4334, aged 19 days, however, it occurs as a long, tongue-shaped structure measuring 0.6 by 0.05 mm. Here it bears well differentiated simple cuboidal epithelium similar to that illustrated by Fischer (1922). The organ is well supplied by capillaries.

In Section 304 of the 6-months fetus there is also seen on the anterior wall of the utriculus a small projection which has been described by Bast, Anson and Wilson as the endolymphatic valve. This structure is well seen in specimen No 4334 also, where it is approximately 0.4 mm. long and is unusually prominent. It lies on the anterior wall of the utriculus and points laterally. It is covered with simple cuboidal epithelium, which becomes columnar on the anterior surface. A capillary supplies the base of the organ. For further reference to these structures see Discussion.

The lateral ampulla, as seen in Section 371, measures in diameter 1.67 mm. by 1.61 mm. The ampulla is cut out, as seen in the model (Fig. 11).

Accessory Crista in Posterior Ampulla.—The posterior ampulla, as has been indicated, presents an anatomic anomaly in that it contains an accessory crista (Fig. 17). Fig. 16 shows a small fleck of the cupola of this crista in the posterior ampulla. This accessory organ has developed on the anterior wall of the ampulla above the level of the normal crista and is independent of it. The structure is complete, being supplied by a branch from the posterior ramus of the vestibular nerve and also with blood

vessels. It is mushroom-shaped, and its main body extends
through seven sections (140 micra) only. This means that it
occurred on but two of the wax plates. These were incorporated
within the block without realizing their significance, and conse-
quently the fourth crista is not evident in the wax model. A series
of projection tracings of the structure was made on celophane.
These corroborated the observation of the independence of the
macula and also brought out the fact that two capillaries take a
parallel course in the perilymphatic tissue just opposite the crista.
The height of the accessory crista is .16 mm., its width .16 mm.
The height of the normal crista in the superior ampulla is 0.258
mm., its width 0.387 mm., Section 304. Fig. 18 shows the cupola
of this crista as it extends across the ampulla. In this view it
presents a pitted appearance. Through the pits can occasionally
be seen cross-sections of "hairs." In Section 304 it presents a
doubly striated appearance, some of the striations going up to-
ward the tip in parallel rows, others curving around the cupola.
With Mallory's connective-tissue stain the cupola stains a brilliant
blue, similar to the reaction of the tectorial membrane which it
greatly resembles. The sensory epithelium of the crista is essen-
tially the same as that described for the macula, the only differ-
ence being that it rises in the crest as it crosses the pit of the am-
pulla, instead of following the contour of the bowl-shaped bottom
of the ampulla. As in the macula a clear space occurs between the
tops of the cells and the cupola. No blue-staining granules were
seen in the cupola, which might indicate calcium.

The saccus endolymphaticus was not observed at autopsy. In
the adult, as Portman (1927) explained, the endolymphatic sac
lies in a "fossette," which is partly covered by an operculum of
bone. At 6-months fetal life this bony operculum has not yet
developed (Fig. 19). By 6-months infant life, however, it has
formed, as may be seen in specimen No. 3920 right, Section 320,
and left, Section 620. Specimen No. 3849 right, Section 350, a
1-month-old child (white), shows the endolymphatic duct emerg-
ing in the middle of the operculum instead of under it.

Fig. 19 shows that the saccus endolymphaticus has an intra-
dural location. The organ measures 4 mm. in an anteroposterior
direction and 1.7 mm. in a supero-inferior direction. Particu-

Fig. 17. Sec. 338. Accessory crista in the ampulla of the posterior semicircular canal.

larly at the level of its inferior surface are seen numerous intradural spaces. Minute diverticula of the main lumen appear to branch off as if creeping between the folds of the convoluted wall. These occur mostly on the postero-inferior wall. They come in very close relation to, if not in actual contact with, the intradural space. A tortuous venous capillary follows closely the

Fig. 18. Sec. 320. Detailed view of normal crista in the superior semicircular canal.
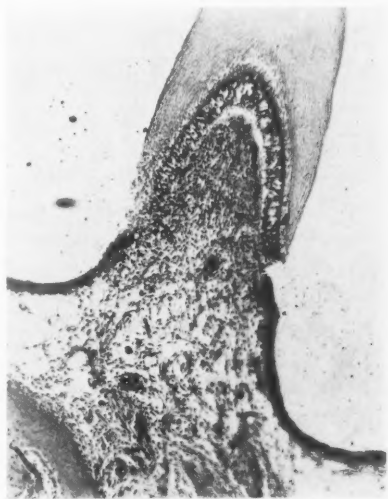
villous-like folds of the saccus. Diapedesis into the dura has occurred near the posterior end of the saccus.

The epithelium of the saccus endolymphaticus is well differentiated. The cytoplasm of many of the cells has a bulging granular appearance and the nuclei are in a basilar position. The epithelium does not present so tufted an appearance as that seen in animal material, yet there is quite an irregularity of the contour of the inner wall. Within the lumen of the saccus may be seen large mononuclear cells, whose nuclei are eccentric. They resemble histiocytes, but their identity cannot be proved by the present staining, so must remain a problem for the future. The cytoplasm of these cells stains pink (H. and E.), but is not granu-
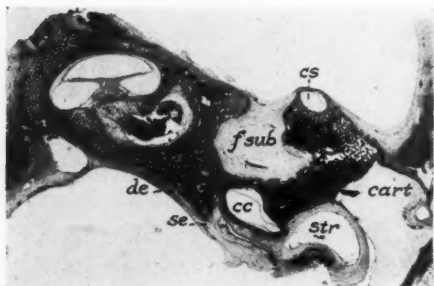
Fig. 19. Sec. 185. The saccus endolymphaticus and the fossa subarcuata: *str.* sinus transversus.

lar. At intervals these cells appear to be working their way through the epithelial wall.

As the ductus endolymphaticus leaves the saccus it is accompanied by a large vein and a very minute capillary plexus. The vein takes a more horizontal course than the ductus and the capillaries follow closely the course of the ductus. The ductus and the vein are seen on the model, Block 8 (Fig. 4). The lumen of this organ by no means occupies the whole of the aqueductus vestibuli, thus leaving ample space for the tortuous course of the accompanying capillary. Its epithelial wall is similar to that of the saccus and is thrown into similar tufted folds. The length of the ductus was estimated to be 1.4 mm. This measurement was taken from the point where the saccus narrowed down to form the

ductus. The diameter of the lumen in Section 203 (Block 8, Fig. 6), is 0.175 mm. by 0.077 mm. Thus it will be seen to approximate the size of the ductus reuniens. The vein accompanying the ductus endolymphaticus measures 0.177 mm. by 0.12 mm.

Perilymphatic System.—The perilymphatic system of the cochlea comprised of the scala tympani and scala vestibuli, has been indicated above. These two form one continuous space, as may be seen in Fig. 16, their union occurring at the helicotrema. The basilar membrane projects into this space, forming the hamulus, and above the hamulus is the terminal portion of the cochlear duct or caecum cupulare. The most important feature of the perilymphatic space in this region is the canaliculus cochleæ. The basal turn of the cochlea, just within the secondary tympanic membrane, shows a decided depression. This depression occurs at the same level as that of the departure of the ductus reuniens from the cochlear duct (Fig. 11). A fact which appears under the microscope but is not evident in the model is that while the perilymphatic space dips into a funnel-like depression of the cochlear canaliculus it does not go through to the subarachnoid space. Close beside this depression is another, over which extends a delicate membrane. This canal does extend the whole way through the bone. Its diameter is 0.2 mm. It contains a few blood cells, although the cells lining the lumen scarcely appear as well differentiated as those of a venous wall. In another specimen, aged 26½ hours, in which sections were cut vertically, two such depressions occur. Both lead all the way to the subarachnoid space. One carries a blood vessel containing blood, the vein of the cochlear canaliculus. The other, the canaliculus cochleæ, contains strands of delicate connective tissue which apparently block the lumen. Running beside these strands is a small capillary. Other specimens in the collection show a rather wide lumen throughout. In many cases of suppurative labyrinthitis this duct is filled with pus cells. As noted above, there is, in young specimens, a fissure (petrosal) running parallel to the canaliculus cochleæ, leading from the fossula fenestræ cochleæ to the dura.

The perilymphatic tissue in the cochlea is represented by the ligamentum spirale. This tissue arises from the endosteum or periosteum (Ruttin) of the peripheral wall of each turn of the

cochlea. On the side toward the modiolus it tapers to attach the
basilar membrane. The main body of fibers of the basilar mem-
brane curve upward around the external sulcus. They send off
radiating fibers into the spiral ligament. As described above, the
ligament supports the spiral prominence, stria vascularis and ves-
tibular membrane of Reissner. The cells of the so-called ligament
are like typical fibroblasts. Near the bony wall they become
smaller and more compact. In some specimens a definite line of
demarcation is seen at this point. Numerous blood vessels occur
in the ligament and many minute tubules containing only mono-
cytes. The caliber of these tubules is such as to admit only one
cell at a time. No pigment was found in the ligament of the 6-
months premature infant (Negro). Occasionally it has been
observed by the author in granular form in this region in older
Negroes.

In each turn of the cochlea the periosteal layer continues from
the spiral ligament back to the modiolus in much reduced form
bearing a rich blood supply. In the basal turn, where the ligament
descends into the vestibule, it becomes continuous with the peri-
lymphatic tissue of the saccular wall.

The cisterna perilymphatica comprises the largest region of
the vestibular division of the perilymphatic labyrinth. It lies just
internal to the footplate of the stapes. The scala vestibuli leads
into it, as may be seen in Fig. 11. Its full extent will be under-
stood if one refers to Fig. 10, remembering that perilymphatic
tissue supports and surrounds the recessus utriculi. Otherwise the
whole area is clear perilymphatic space. The depth of the space
is 18 mm. From the footplate of the stapes to the body of the
utricle is a distance of 0.88 mm. The anteroposterior measure-
ment, as seen in Section 344 (Fig. 9), is 3 mm. In Fig. 9,
Section 304, the area on both sides of the lower part of the utricle
is for the most part free from any perilymphatic tissue.

The saccule and utricle are supported and held in place by the
delicate areolar perilymphatic tissue. So also are the cristæ ampul-
lares and the semicircular ducts of the canals. This tissue is not
so well organized as the spiral ligament of the cochlea. Some
observers, Alexander, Ruttin, etc., have, however, found certain
strands of this tissue to be constant enough to bear localizing

names. Ruttin, for example, locates the macula neglecta in the
"inferior septum." A rather dense mass of perilymphatic tissue
appears between the utricle and horizontal ampulla (Fig. 9),
giving a form identical with that of a crista. It bears no special-
ized epithelium, however. An extension of this forms the eleva-
tion known as the pyramis vestibuli. In the construction of the
model these features were sometimes temporarily confused with
true cristæ. The perilymphatic tissue of the canals, although wide-
ly meshed, leaves no space at this age absolutely free in the
canals, such as the cisterna of the vestibule. In addition to sup-
porting the endolymphatic system it also supports the vascular
system. In tangential cuts of either the cochlea or saccule an
arcuate arrangement of capillaries is seen in the perilymphatic
tissue. In the canals the blood vessels are borne along the oppo-
site side from the semicircular duct.

True histiocytes can surely be differentiated, at the present
time, only by vital staining with specific dyes. Nevertheless, large
amoeboid cells are occasionally seen in the perilymphatic tissue,
as for example, in Section 300, below the region of the macula
neglecta.

Nerve Supply.—The eighth nerve consists of two elements, the
cochlear and vestibular. As described in the study of the capsule,
the internal auditory meatus lies relatively high on the capsule
in the 6-months fetus, and its skeletal division into superior
and inferior regions is incomplete. The cochlear nerve enters
the cribriform base of the modiolus and distributes its spirally
disposed fibers to the different levels of the bony groove which
carries the spiral ganglion. The spiral ganglion cells are small
and contain little cytoplasm, and therefore seem to be immature
in the 6-months fetus. From the spiral ganglion the fibers pass
out in the fenestrated area between the two plates of the osseous
spiral lamina, to the organ of Corti. It is clearly seen that the
fibers from the basal turn of the cochlea branch off from the main
axis very early in the apical course of the nerve. A rich blood
supply is seen around the spiral ganglion and among its cells.

The peripheral ganglion for the vestibular nerve lies in the
internal auditory meatus. Like the spiral ganglion, the cells of
the vestibular ganglion, as shown by their small size and the

paucity of cytoplasm, are immature. In adult specimens the vestibular ganglion cells appear larger than the cochlear cells.

From the vestibular ganglion the branches are distributed to the divers parts of the vestibule. The superior branch follows closely under the facial nerve and is distributed to the superior and horizontal ampullæ and to the recessus utriculi. The bony canals for these branches may be easily followed in the model Blocks 4, 5 and 6. The branch to the posterior ampulla is well illustrated in Fig. 6 as it makes an almost right-angled turn to enter the foramen singulare. A branch of the posterior ramus supplies the accessory crista described above. Fig. 7 shows the saccular branch of the vestibular nerve approaching that organ.

Blood Supply.—The division of the cochlear artery into two branches may be studied in Block 9 in the model. One branch penetrates deeply, following the cochlear nerve to the modiolar axis. The other follows the nerve fibers to the basal turn and sends off a branch to the saccule. No attempt to follow the vessel farther was made. In the vestibule a rich vascular supply is noted under each macula and under the cristæ. Under the macula the vessels run parallel to the surface.

In the cristæ two main vessels appear to run parallel with the surface of the crest along the long axis. One of these is along the base, the other nearer the surface. Immediately under the epithelium minute capillaries run apparently at right angles to the long axis of the crest. As stated above, the vessels for the nonampullated part of the canals follow the proximal curve of the canals while the semicircular duct follows the peripheral curve. In the posterior canal two vessels are seen. They send occasional capillaries across toward the semicircular duct.

In the cochlea the branches of the artery are surrounded by large perivascular spaces. The vessels escape from the modiolus through apertures in the bony modiolus, into the perilymphatic tissue of the cochlea. A branch is seen following along the basal border of the osseous spiral lamina. No vessel could be seen immediately under the organ of Corti, as occurs in rabbits and other animals, and has been described for man. Numerous vessels are seen in the spiral ligament, and the structure of the stria vascularis indicates that a series of parallel vessels run around the

cochlea in this region. As stated above, a tangential cut of the spiral ligament shows the capillaries arranged in an arcuate manner. The caliber of these vessels is approximately that of the diameter of a red blood cell. A cell larger than this distends the wall markedly.

No pigment was observed in the perivascular or perilymphatic tissue of the inner ear of the 6-months fetus (Negro), although it was observed in granular form in the cells of the epidermis of the external auditory meatus. In the perilymphatic tissue supporting the crista of the superior canal a faint brownish tinge appeared in the cytoplasm of the cells. This was not definite enough for a positive statement of the presence of melanin.

III. DISCUSSION.

The study of the history of the anatomy of the ear is a subject so specialized that space is not generally given to it in histories of anatomy. In spite of the fact that Politzer, Retzius, Siebenmann et al. have meticulously recorded the history of the subjects of which they treat, their information has not gotten before the general historians of anatomy and has not been incorporated in their works. Personal prejudice and national feeling have evidently in some cases prevented posterity from knowing important and interesting facts. On the other hand, time alone can sift the significant from the insignificant, and an effort to establish priority in a period contemporary with the author is often useless. Politzer in his comprehensive two-volume work fails to more than mention Corti. Interested readers are just beginning to realize that ideas similar to those expressed in the Helmholtz theory of resonance had previously been generated in different countries in the minds of various investigators, as Valsalva, du Verney and Cotugno. While establishing priority is an interesting and important academic contribution to our knowledge, of still greater importance is a full appreciation of the zealous spirit which dominated the early investigators.

Cavum Tympani.—The observation of the inferior location of the tympanum in relation to the otic capsule in the 6-months fetus is in keeping with the statement of Keibel and Mall and with the observations of Hammar, to whose work the preceding authors

refer. Keibel and Mall state that "In the sixth month the os petrosum rotates around its long axis toward the outer side, the cupola of the cochlea becoming depressed and a temporary depression of the tympanic cavity is thereby produced, so that its walls again assume an almost horizontal position" (p. 276).

Hammar (1902), in making a study of the origin of the thymus found it necessary to construct models showing the development of the anterior part of the gut. For this purpose he made use of twenty-six specimens ranging from four weeks to ten months. In the paper here cited he gives a careful description of the development of the middle-ear cavity. His fetus and model XIX, 150 mm.—six months—correspond closely to the one described in this paper. Of his specimen he says: "Ist diese Höhle wieder in eine beinahe horizontale übergetreten. Die mediale Wand sieht jetzt mit ihrer Aussenfläche vorzugsweise nach oben die laterale vorzugsweise nach unten." On the other hand, a statement is made further on that the tegmen tympani has an almost horizontal position, a feature not corresponding to the observations of the present writer nor, if correctly interpreted, to the other relationship described.

Since only one stage (6-months fetus) was modeled in the present study, no statement can be made here as to how the cavum tympani acquired this inferior position. However, from observations of serial sections of the 3-months fetus, where the whole head was sectioned and therefore relatively good orientation was obtained, the cavum tympani appears to have a decidedly inferior position at three months also. In speaking of the further development of the tympanum from seven months on, Hammar states that the shift in position is accomplished not by rotation but by resorption of bone. The present writer, however, is forced to the conclusion that there must also be a rotation in order to bring all related parts into the adult position.

The measurements made upon the ossicles indicate that they are approximately as large as in the adult, an observation in accord with that of other investigators.

Muscles.—Broman states that the tensor tympani appears at the end of the second month. He finds that it is connected at its

distal end with the veli palatini and with the tensor veli palatini, a connection which is dissolved at the end of the third month. In the present study the tensor tympani was well developed, and no connection was observed in the 3-months fetus.

Nervus Facialis.—The exposed condition of the facial nerve as it occurs in the late fetus and early infancy is of phylogenetic, ontogenetic and clinical importance. Here we have this important nerve crossing the tympanum in an open gutter. Dr. R. J. Terry finds that the facial nerve never actually traverses the otic capsule but only appears to do so. He believes that in reality it follows a foramen formed by the meeting place of the otic capsule and the suprafacial commissure, a derivative of the basal plate of the chondrocranium. He finds this to be true for Chrysemys and Necturus. Dr. Terry states that the acoustic meatus is formed chiefly by the growth of the suprafacial commissure, resulting in a close relationship of facial and acoustic nerves and apparently burying the former in the otic capsule. In Necturus he finds that a secondary floor grows inward to separate the hyomandibular part of the facial ganglion from the auditory chamber. He states that it is the rule in mammals for the facial nerve to traverse an open groove in the tympanic wall and that such is the case in adult Felis domestica.

The present writer presents two exceptions to this rule. The primates Pithecus (Macacus) rhesus and man. Four pairs of monkeys studied showed no aperture in the bony facial canal. In the adult man normally the canalis facialis is a closed canal with occasional apertures for the passage of minute vessels. In late fetus and young infants, however, and very occasionally in adults, sizable apertures may occur. These are called "dehiscences" in the clinical literature. With no desire to confuse or increase terminology the word "aperture" seemed to the present writer more properly applicable than "dehiscence." "Dehiscence" comes from the Latin word *dehiscere*, meaning to gape. "Dehiscence" is defined by Stedman as a splitting or bursting open. Dorland defines it as the act or process of splitting; Webster: (1) A gaping open, (2) Act of opening along a definite line to discharge contents, as a capsule or pod. If the word connotes an active process of splitting or bursting open, it is certainly not at

all applicable. If it connotes a more or less haphazard gaping
caused during the process of pneumatization, as suggested by
Wittmaack, it is again not applicable. Nor is it applicable if it is
thought of as a sloughing off from disease processes. These aper-
tures are not random occurrences but have an ontogenetic sig-
nificance in that they indicate how completely the facial nerve
has become enclosed by the otic capsule, since the early fetal days
when it crossed the tympanic cavity completely exposed (three
months). The present study has shown that the largest and most
constant aperture occurs where the facial nerve initiates its de-
scending course just posterior to the oval window. Perusal of the
literature will show that when a stapedial artery persists it enters
the facial canal at this point. No examples of persistent stapedial
artery occur in the collection of human material in this laboratory.

These apertures have also a phylogenetic significance. A study
of animal material in this connection was revealing. Adult white
rat showed a very large opening posterior to the oval window
through which passed the stapedial artery. This vessel, after fol-
lowing the facial canal for a distance, becomes continuous with a
vessel within the cranium.

Tandler also gives an excellent series of plates illustrating the
course of the stapedial artery in the white rat and the series of
changes through which the aortic arches pass in the course of
embryonic development. He states that the stapedial artery arises
from the internal carotid, passes through the middle ear and
forms the supra-orbital, infra-orbital, lacrimal, ethmoidal and
muscular branches. Further observations have been noted above.

Tandler (1902) lists fourteen names of those who have con-
tributed to our knowledge concerning the stapedial artery. These
include (1) Salensky, 1880; (2) Fraser, 1882; (3) Gradenigo,
1887; (4) Noorden, 1887; (5) Rabl, 1887; (6) Staderini, 1891;
(7) Dreyfuss, 1892; (8) Baumgarten, 1892; (9) Siebenmann,
1894; (10) Jakobi, 1895; (11) Zondek, 1895; (12) Hegetsch-
weiler, 1898; (13) Broman, 1899; (14) Grosser, 1901.

It is impossible to state at this time whether or not the stapedial
artery instead of completely disappearing at the end of the third
month (as stated by Broman) merely becomes reduced to capil-
lary size, supplies the mucous membrane of the region, and enters

the facial canal. Cases of persistent stapedial artery in man, reported in the literature, would seem to bear out the latter possibility.

The clinical significance of these apertures is obvious. Temporary facial paralysis occurs with otitis media. The persistent apertures of the bony canal readily explain the possibility of an entering infection. An entering blood vessel (arteriole) still further completes the picture for the spread of infection, edema or increased pressure. According to Wittmaack (1926), Kerrison (1930) and Politzer (Ballin) (1926) facial paralysis occurs most frequently in inflammatory processes of the middle ear of the newborn but may also occur in later life. Wittmaack asserts that the pathologico-anatomic cause of this is not established, but he attributes it to pathologic dehiscences occurring in the course of pneumatization. The present writer believes the cause is purely anatomic and not pathologic except in a secondary way. It is not due to dehiscence nor even to "spontaneous" dehiscence. The true condition of affairs is that the bone had never been developed in the particular region involved. The exposed facial nerve is a developmental condition, a phylogenetic feature which once was the point of entrance of the stapedial artery.

Tuba Auditiva.—From the observations upon the auditory tube one may state that the tube is patent from the early stages of its development and never contains mesenchyme as is found in the tympanum. The epithelium of the tube becomes differentiated in the third month of fetal life. It does not differentiate so early as the lingual or nasal epithelium. By the fourth month of fetal life the epithelium is further differentiated in different parts of the tube.

A constriction appears in the course of the tube as early as the third month. This does not appear to be peculiarly related to the origin of the tubal cartilage as in the adult, since the cartilage has not yet appeared. Cartilage first appears laterally and near the pharyngeal end in the fourth month. By the sixth fetal month the cartilaginous tube extends to the level of the cochlear apex. This condition is maintained until after birth. No bony tube was observed in a ten-day infant. It was well formed, however, at two and one-half months.

In the fourth fetal month a tortion of the auditory tube was observed. This occurred in such manner that whereas the long axis of the tubal lumen lies in a horizontal plane at the pharyngeal end, it becomes vertical farther up the tube.

Krause observed that the cartilage of the auditory tube first appeared in the fourth month and stated that it has no relation to the visceral arch. Some authors, however, connect it with the spiracle of lower forms. Hammar states that the tube first acquires its sickle shape in the sixth month of fetal life, at which time it becomes significantly longer. He observed a slight spiral turning which we have noted as present in the fourth month.

Mesenchyme.—Much discussion has arisen as to the time of disappearance of the mesenchyme in the middle ear. In the present study, made upon specimens classified according to age, it is clearly seen that mesenchyme was not observed in specimens older than 13 months. The submucosa may remain thickened, however, until about the eighteenth month.

Following infection there may develop a delicate areolar tissue which is sometimes taken for mesenchyme. True mesenchyme may be distinguished from this recently formed fibrillar connective tissue by a detailed study of serial sections. If the delicate stroma exhibits large, full nuclei and is completely homogeneous with the submucosa at every level in which it appears, it may then be considered as mesenchyme. If, on the other hand, at some levels it shows the surface of the mucosa (as indicated by the arrow in Fig. 22) extending through the tissue, this means that the young granulation tissue or fibrillar tissue, or a polyp-like structure has grown out from some irritated region and is encroaching upon the neighboring mucosa.

A second point of differentiation lies in the fact that the very delicate processes of mesenchymal cells are free to grow in any direction and spread an even matrix. The processes of fibrillar connective-tissue cells and scar tissue, on the other hand, have a definite polarization. Their direction of growth is controlled by the location of the infection which they are attempting to wall off.

Both types of tissue arise from the same primary cell. It is therefore to be expected that the two will be similar. Confusion may arise when infection occurs in infant ears in which the

Fig. 20. Osteoblasts in peri-
osteal bone.
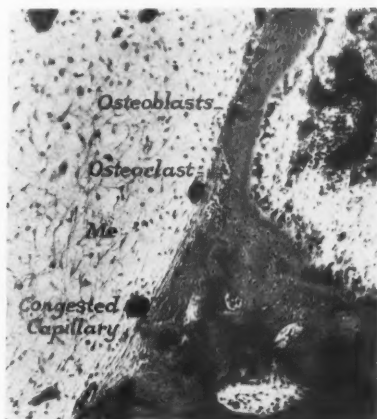
mesenchyme has not yet been completely resorbed. Specimen
No. 3951, from a 5-year-old child, presented some difficulty in
deciding the type of tissue in the antrum and epitympanum until
it was observed that columnar, and in places ciliated, epithelium
lined all the tissue surfaces. Mesenchymal surfaces are never cov-
ered by columnar epithelium.

Since the above observations were made a publication by L.
Singer has appeared further substantiating the view that mesen-
chyme may be differentiated from tissue growing in response to
stimulation from infection. Singer states that if spindle-shaped

Fig. 21. Osteoclasts.

cells are present the tissue is scar tissue and not mesenchyme.
M. Schwarz (1931) has made an excellent study of mesenchyme
in 100 fetuses, ranging from three months to full term. He
showed that mesenchyme alters in structure from the third to the
tenth month, gradually assuming the character of loose connective
tissue, and its cells becoming more typical fibroblasts. Actual
pneumatization of the mesenchyme, according to his observations
also, is a much earlier and more rapid process than previously
supposed.

Fig. 22. Specimen No. 3903.
Right. Sec. 594. Fibrillar con-
nective tissue, *sct*, in a parant-
ral cell of an 8-year-old boy.

Keibel and Mall (Ed. 2, p. 278) state that areolar tissue
(mesenchyme) disappears only after birth, the tympanic cavity at
that time almost or completely lacking a lumen. A further dis-
cussion is given of the forensic importance of the condition of
the middle ear at this age.

This importance should be stressed with caution. In all em-
bryonic (two months) and fetal material observed by the present
writer, a well-defined lumen was present in the middle ear lead-
ing from the auditory tube and appearing first along the mem-
brana tympani. This original auditory tubal lumen never contains
mesenchyme. Hammar states that in the third month the forma-

tion of mesenchyme is at its height. This is quite true, but at the
same time the lumen of the auditory tube has progressed pos-
teriorly along the membrana tympani. Hammar also states that
for a time in the sixth month the lumen is obliterated. This does
not correspond to the present observations. The condition de-
scribed by him may possibly be explained by the presence of a
pathologic condition in which granulation tissue might have ob-
structed the lumen. In all specimens observed by the present
writer cavity formation undoubtedly appears to be a constantly
progressive process.

Fig. 23. Specimen No. 3903,
Left. Sec. 336. Cystic scar
tissue in the niche of the round
window.

The persistence of this mesenchyme readily explains why in
many premature infants no drainage of pus occurs after myrin-
gotomy. The "flat" or horizontal position of the drum membrane
makes the location of the posterior inferior quadrant quite dif-
ficult. If the posterior superior quadrant should be cut, drainage
could not be expected at this age. On the other hand, it is readily
seen why an incision here should yield free blood, as the mesen-
chyme is highly vascular.

If mesenchyme were observed in the adult with no evidence of
concomitant or preëxisting infection, then and then only might

we assume that this embryonic tissue persisted in the tympanum until adult life. The writer has observed no such cases.

Membrana Tympani.—The membrana tympani had begun to develop in material obtained late in the second month of embryonic life. The annulus tympanicus at this early age had already begun to ossify, showing its unmistakable bony horseshoe shape in cross section. In the sixth month of fetal life it is well developed. Its blood supply, as pointed out by Ormerod, comes from both an external and an internal source. The external supply, according
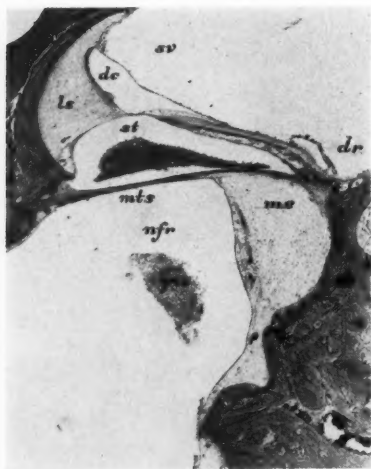
Fig. 24. Specimen No. 4439. Left. Sec. 270. Mesenchymal remnant in niche of round window of 15-day infant.

to him, is from the deep auricular branch of the internal maxillary and the internal supply comes from the tympanic plexus.

Pars Petrosa.—Inferiorly the sphenopetrosal fissure, when no longer covered by dura, opens into the cavity of the middle ear, the roof of which is undeveloped at this age. The posterior fissure (fissura petro-occipitalis), when uncovered by dura, leads at six months fetal life into the fascial spaces of the neck in the region of the jugular vein.

The formation of the bone in the auditory capsule still remains a subject of great controversy. Bast (1930) believes that all three

types of bone in the otic capsule form simultaneously. He bases his argument on the study of a 5-months fetus. Bast has made a thorough study of the ossification of the temporal bone, locating the numerous centers in the capsule. In a 6-months fetus all three types have already appeared and the centers of ossification mostly coalesced. The writer differs in one point with Bast. She believes the enchondral bone to be a trabecular formation and not an island formation. Periosteal bone apparently matures more quickly, once started to develop, than does enchondral bone.

Weber's time-phase thus enters into the explanation of the appearance of the different layers. In a 6-months fetus all three types have already appeared. The enchondral type is the last to ossify.. By some authors this has been taken to mean that this layer represents the primitive cartilaginous capsule of the primordial skull. As is well known, its osteoid condition remains late in life, even extending into adult life. This layer is generally described as composed of "cartilage islands," but as above stated, when their course is traced they prove to be tortuous trabeculæ of osteoid tissue. Perhaps they are tortuous merely because they are developing between two already ossified layers of bone and following a common biologic law (as does the digestive tract) are forced to curve as they develop; or perhaps their incomplete ossification during the time of rotation of the otic capsule may explain this phenomenon.

Werner (1932) considers that the endosteal layer of bone is lacking at the middle point of the curve in the developing semicircular canals, and that the blood vessels are directed concentrically toward the canal. He observes that irregularities are especially frequent in the ampulla of the posterior canal. M. Meyer (1931) finds that the endosteal layer is lacking on the periphery of the canals and that it is here that the cochlea and canals enlarge. According to this author, giant osteoclasts occur here, while on the inner wall of the canals the "cartilage rest-cells" occur, which are resistant and no change in shape occurs. He states that where notched "cartilage rest-cells" encroach upon the canal it may assume a kidney shape instead of a round shape.

The region in which enchondral bone forms is so open that one almost feels that the endosteal capsule of the cochlea must be

floating in a bed of marrow into which are being injected a few
of these enchondral trabeculæ. These marrow spaces may be in
direct continuity with either the tympanum or the labyrinth in
young specimens.

That sanguefactive marrow occurs in the cranial bones of the
head has long been known. But the persistence of this type of
marrow in the incompletely pneumatized temporal bone of infancy
and early childhood has not been generally recognized. It is well
illustrated in Henke and Lubarsch (1926), however. This con-
dition is to be expected since we know the diploe of the skull con-
tain sanguefactive marrow. A premature infant has usually no
parantral cells pneumatized. Pneumatization of the bone is incom-
plete until puberty or later. Until that time hemopoietic marrow
exists in these cells. This type of marrow persists until an even
later period in the petrous apex. It is replaced by a more fatty
type of marrow where pneumatization fails to occur.

Considering the fact that the temporal bone is formed in part
from the primitive chondrocranium, it is not surprising if rem-
nants of cartilage may be found rather late in the course of
development. Such has proved to be the case. It has long been
recognized that the basal cartilage filling in the lacerate foramen
remains cartilage throughout life. This is a remnant of the
chondrocranium (Morris' Anatomy). Just as certain areas of the
cavum tympani have persistent mesenchyme, so certain areas of
the bony capsule have persistent centers of cartilage. Expressed
in other words, these areas succumb to ossification later than does
the rest of the otic capsule.

Pars Petrosa.—The rotation of the otic capsule is a funda-
mental process in the course of development. But rotation is not
confined to the otic capsule. The whole petrous bone is involved,
including the tympanum and the eustachian tube. The vertical
position of the stapedial crura of the 6-months fetus, as com-
pared with their horizontal position in the adult, evidences this,
as does the shifting of the plane of the drum membrane from the
horizontal to the more vertical position. This shifting may be
readily observed in any series of fetal and infant skulls of differ-
ent ages. The "flat" position of the drum membrane, as described
by hospital internes in recording their observations on the prema-

ture infants, is undoubtedly explained by the early anatomic po-
sition of the drum membrane. This rotation is not often discussed
in the literature. Broman mentions it when discussing the facets
of the developing malleus. Hannover discusses it, but the present
writer is handicapped by inability to read the Danish.

It is interesting to note that Hrdlicka finds the absence of the
external auditory meatus in early American skulls occurring only
in the right side. The anatomic anomalies recorded in this paper
are unilateral and on the right.

Fossa Subarcuata.—The great extent of the subarcuate fossa
in early infancy is of considerable significance clinically. A route
of infection by blood stream from middle ear to meninges, and
vice versa, by way of the subarcuate fossa, has long been recog-
nized. From the present study it now seems possible that infection
by direct extension through the dura in a fossa unprotected by
bone is a possibility in many cases because of anatomic conditions.
Fortunately, the dura is normally quite resistant to infiltration.
Whether or not it permits toxic fluids to penetrate it with greater
ease than it permits cellular infiltration, cannot be said. In each
of the cases in which the subarcuate fossa could be traced through
to the mastoid antrum, the individual died of meningitis and
showed otitis media. From the pathologic point of view, it is
interesting to note that in no case observed was there a pathologic
infiltration of the dural tissue leading from the cranial cavity
into the subarcuate fossa. But in all cases the small vein and
artery showed evidence of thrombi and congestion. The fact
that the connection with the antrum was detected more frequently
on the left and also that there seems to be a tendency for the right
side to close earlier than the left, points to a possible anatomic
difference in the two sides. It is recognized that the number of
cases observed is too few to permit drawing any conclusion in
this matter. Quite frequently there is but a fraction of a millime-
ter of bone separating the fossa from the mastoid antrum.

In view of the statement occurring in Huber's edition of Pier-
sol's Anatomy (1930), page 1509, "The right side of the head
has been said to be more frequently affected by intracranial
sequelæ . . ." it is of interest that in four cases observed the
subarcuate fossa remained patent on the left side only. The

accumulation of data on this point should be valuable in determining the possibilities. The possible influence of the larger right lateral sinus must be considered in this connection.

Fossula Petrosa.—Froriep has described in 8 to 12 mm. embryos the glossopharyngeal nerve approaching the caudal ventral border of the ear vesicle and passing on to come in contact with the epidermis of the pharyngeal cleft. The course is described as diagonal. This condition found in earlier mammalian embryos explains the diagonal fossula pretrosa (Hyrtl) occurring in the 5-months premature infant and frequently seen postnatally. Its diagonal course is parallel with the diagonal course of the cochlear aqueduct, with which it is in no way connected. The patent fossula might obviously afford a route of direct extension of infection from a suppurating middle ear.

Osselets.—Augier has described the presence of a suprategmenal osselet as well as a supracochlear osselet. He believes these to be remnants of the suprafacial commissure of the primordial skull and that they soon become incorporated in the developing cranium and their identity lost. He refers to the theory of Kaufmann that the tegmen tympani is a new acquisition since the stage and type of the reptilian cranium.

Membranous Labyrinth, Cochlear Division.—It is interesting to note that the expansion of the cochlear duct is greater in the lower middle and upper basal turns than elsewhere. Wrightson found such an expansion in the width of the human basilar membrane and spiral lamina. Guild and his co-workers found the greatest number of ganglion cells occurring in the upper basal and lower middle turns. Lorenti found the largest nerve bundles in this region.

Measurements made by the present writer on a series of sections of tangential cuts of the cochlea show that the duct appears to be expanded and contracted within short distances, in addition to undergoing a gradual change in size from coil to coil. These findings must be the measured indication of the wave of vibrations passing along the cochlear duct. The observations are interesting in view of those of Wrightson on the basilar membrane. He states that the curvature of the basilar membrane alters in every phase. It is flat, concave, and convex in turn.

It seems somewhat surprising that the height of the organ of Corti should increase in the apical turn, thereby presumably increasing the load on that part of the basilar membrane.

Vestibular Division.—It is difficult to know whether the relatively small macula of the utricle is merely a stage in development or an anomaly. It is probably the former, for a study of older specimens seems to indicate a more extensive macula than those present in the fetal specimens. Benjamin (1913) describes a "fourth crista" in a 5-months fetus similar in structure and location to that described in this paper. He considers it the same as the crista neglecta. Sakai (1922) made a study of twenty fetuses and sixty specimens ranging from birth to adult. In this series he reports the occurrence of the "macula neglecta" in 95 per cent of the fetuses, in 90 per cent of the children, and in 85 per cent of the adults. Fischer (1922), in reviewing the history of the subject, says that Alexander was the first to recognize that the macula neglecta does exist in higher mammals. Fischer based his own conclusions on a model. He believes that these peculiar epithelial formations can occur in a series of four possible groups. Stütz (1911) had also worked on the subject by the aid of a model. In one of his excellent illustrations he definitely shows one part of the rudimentary crista appearing in the neighborhood of the entrance of the endolymphatic duct into the vestibule. Ruttin described a crista (macula) neglecta on the inferior septum of perilymphatic tissue. He bases his argument that this really is an extra crista on the fact that pigment occurs in it, as stated above.

Endolymphatic Valve.—From its location in specimen No. 3887, described in detail in this paper, it scarcely seems possible that the "endolymphatic valve" could function as a valve. In other specimens, however, the writer has observed it appearing as a flap-like valve quite as described by Bast (1928), Anson and Wilson (1929). Rudolph Krause (1906), in his Entwickelungsgeschichte des Gehörorgans," describes and illustrates (Fig. 110) a connective-tissue fold which penetrates from above between the utriculus and sacculus, occurring exactly at the mouth of the ductus endolymphaticus.

It occurs to the writer that the endolymphatic valve described by the American authors may be really part of the same structure as that described by Krause, Stütz, Fischer, Sakai, Ruttin, Benjamin et al. In other words, the medial end of the rudimentary macula or (crista) neglecta may be taking upon itself a new function, that of a valve, or it may merely represent the vestigial end of an organ which once functioned in the lower part of the utricle. Joseph Fischer reports a third macula in the utricle which divides that sac into two distinct parts. He observed a nerve supply to this macula. He inclined to the opinion that it is atavistic.

Saccus Endolymphaticus.—The precise location of the saccus endolymphaticus in regard to the meninges is sometimes misunderstood. It is intradural, as described by Retzius, and not subdural or epidural. Were it epidural it would be seen when the autopsies are performed; were it subdural it would be seen when stripping the dura from the temporal bone, a procedure which should not be done if one expects to see the organ in the microscopic sections. Usually the saccus is never noticed at autopsy. Occasionally, however, it is so distended that a flabby area appears in the dura just posterior to the internal auditory porus. The saccus is always in close relation to the sigmoid sinus. An actual connection of the saccus cavity with the intradural spaces would not be incompatible with the findings in comparative anatomy Dempster (1930) has described a very extensive endolymphatic system in the amphibians. In Cryptobranchus, according to this author, the system is expanded into the cranial cavity on the two sides. In some animals the sacs form a ring around the brain.

According to Guild, the escape of the endolymph in the guinea pig is through the pars intermedia of the saccus. The pars intermedia of man has not been described in the literature as yet, but the possibility arises of that region being comparable to what has here been described as the posterior inferior part of the saccus. The present writer observed a very intimate location in space between the choroid plexus and the walls of the developing endolymphatic sac in the embryo rabbit. Moreover, the cellular structure of these organs is highly similar. It is difficult to tell whether the cells which appeared to be working their way through the epithelial wall are not really engorged with secretion which they

are about to discharge. On the other hand, the author has observed such cells frequently free in cases of labyrinthitis, in the endolymphatic sac and duct. Relatively rarely are polymorphonuclear cells found in this region even in suppurative labyrinthitis. Arguing on the basis of Weeds' observations in regard to the origin of the cerebrospinal fluid, perhaps we may find that the endolymphatic fluid is not secreted by any glandular organ, as described by Shambaugh, but filtered from the blood. As to whether such filtering occurs in the stria or in the walls of the endolymphatic sac, or in both, only further detailed experiments will show. If Guild's work on the direction of flow of the endolymph is proved physiologically to be normal, the filtering probably occurs in the stria.

The study of the model in this specimen reveals no anatomic reason why perilymph from both cochlea and vestibule might not escape from the cochlear aqueduct. The stria might then be considered the source of origin for endolymph in the cochlea, the saccus for the vestibule.

Streeter (1916) has given an excellent description of the vascular plexus which surrounds the saccus and ductus endolymphaticus. This is based upon an injection preparation. Streeter considers the plexus to be venous. In the material (specimen No. 3887) described in the present paper it is true that the capillaries following the villi of this organ are filled with blood, further substantiating the possibility of their being venous. However, the vessels have a constant round cross-section and a well-defined endothelial wall. This wall, in the very minute branches, possesses a much more definite structure than the wall of the much larger vein accompanying the aqueduct but coursing in its own bony channel.

Canaliculus Cochleæ.—The canaliculus cochleæ in dried preparations presents a slightly curved and downward course. It usually opens with a wide flare beneath the petrous bone. Although a discussion of physiology is not the province of this paper, it may be said that the whole course of the duct in the adult suggests an outflow from the cochlea. According to Karbowski (1930) and Muerman (1928), mammals that carry the head higher than the rump, as man, monkey, and camel, have a nar-

row canaliculus cochleæ. Mammals that carry the head lower than the rump have a wide open cochlear canaliculus. These authors found that the injection of india ink into the subarachnoid spaces appeared along the canaliculus and the vessel, also in the peri-lymphatic spaces of the cochlea and vestibule, in the marrow and along the tympanic nerve. Karbowski found the space into which the canal emptied to be directly continuous with that around the ninth nerve. The possibility of this duct draining the vestibule also has been suggested above.

Cisterna Perilymphatica.—Retzius describes the cisterna peri-lymphatica as of oval shape, measuring about 3 mm. by 3 mm. According to Alexander (1922), in many cases the cavity of the cisterna perilymphatica is completely isolated by the perilymphatic tissue. He identifies three zones in the structure of the perilym-phatic tissue, an endosteal, subepithelial, and an intermediate layer. As early as in the fifth month of fetal life he differentiates three distinct septa (previously called "ligaments" in the litera-ture). These are: (1) a septum extending from the posterior (sagittal) ampulla to the upper outer region of the utricle; (2) a septum from the posterior border of the crista vestibularis to the posterior circumference of the vestibule, supporting the re-cessus utriculi and the horizontal ampulla; (3) a bordering mem-brane or septum occurs over the mouth of the aqueductus ves-tibuli. This membrane is pierced only by the ductus endolym-phaticus. Alexander further believes that the perilymphatic tis-sues serve as a protective apparatus, hindering the perilymphatic waves, which he believes activate the organ of Corti, from stimu-lating the labyrinth. In like manner he believes the very narrow ductus reuniens prevents stimulation of the organ of Corti by movement of the labyrinth.

Rüdinger (1888) had designated the "ligaments" of the peri-lymphatic tissue as the radial ligament of the saccule, the lateral and medial of the utricle. To these Ruttin (1921) adds an inferior ligament for the utricle. It is on this inferior ligament or septum that this author finds the crista or macula neglecta.

Pigment.—The exact time of appearance of melanin in the labyrinth has not yet been determined. As reported elsewhere (Wolff, 1930), the author has observed it in the labyrinth of a

Negro stillborn, believed to be four days premature. We do not know the significance of this substance. Some have considered it a form of waste product; others, a heat regulating membrane. Neither explanation quite satisfies the questions which its presence arouses. One may expect to find it in all Negro material after birth. It is interesting to note that Ruttin considered the presence of pigment in the base of a structure he had designated as *macula neglecta* to be proof of the fact that it was a true macula.

Melanin as it occurs in the labyrinth is evidently in structure similar to the more deeply lying melanoblasts or chromatophores discussed by Cowdry as the second site of occurrence of melanin in the skin. He states that they are in finely branched processes between the epithelial cells and that it is not even certain if they are actually of cellular nature.

IV. SUMMARY.

Early epochal investigations of the anatomy of the ear are reviewed. A routine method for procuring and preparing microscopic sections of the petrous portion of the human temporal bone is given. Detailed gross and microscopic observations of the auditory mechanism of a 6-months premature fetus of the American Negro are made. A wax-plate model of the labyrinth constructed from photographic negatives of serial sections of this specimen is described.

The study revealed that at this age the tympanum is inferiorly placed in relation to the pars petrosa; that the facial nerve lies in an open groove; that pigment has not yet developed in the inner ear, although present in granular form in the skin of the external auditory canal. Certain anatomic variations are noted, such as an accessory crista in the posterior ampulla, irregularities in the contour of the superior and lateral bony semicircular canals.

Comparative tables are presented covering over forty cases, ranging from late embryo to adult. Data are given confirming previous work and showing that: (1) postnatal apertures, so-called "dehiscences," in the bony facial canal have a phylogenetic and ontogenetic explanation; (2) embryonic mesenchyme is resorbed in the tympanum normally by the middle of the second year; (3) the subarcuate fossa may penetrate to the antral cavity. A phylogenetic explanation for the endolymphatic valve is offered.

V. KEY TO ABBREVIATIONS USED IN TEXT AND FIGURES

a—antrum
aap—accessory ampulla
ac—aqueductus cochleae
acl—ampulla canalis lateralis
acp—ampulla canalis posterior
acs—ampulla canalis superior
asc—anterior stapedial crus

bc—basal coil of cochlea
bj—bulbus jugularis
bs—basis stapedis
bv—blood vessel

c—cochlea
cal—crista ampullaris lateralis
cart—cartilage
cc—crus commune
cf—canalis facialis
cht—chorda tympani
cl—canalis semicircularis lateralis
clm—canalis semicircularis lateralis
    medial limb
cp—canalis semicircularis posterior
cps—cisterna perilymphatica
cos—caput os stapedis
cra—accessory crista
crp—canal for ramus nervi posterioris vestibularis.
ct—cavum tympani
cs—canalis semicircularis superior
csl—canalis semicircularis superior
    lateral limb
csm—canalis semicircularis superior
    medial limb
cu—cupola
cvs—canal for nervus vestibularis
    superior

dc—ductus cochlearis
de—ductus endolymphaticus
dr—ductus reuniens
dsl—ductus semicircularis lateralis
dsp—ductus semicircularis posterior
dss—ductus semicircularis superior
du—ductus utriculo-saccularis

e—epitympanum

faf—fissura ante fenestram
ffc—fossula fenestræ cochleæ
ffr—fossula fenestræ rotundæ
ffv—fossula fenestræ vestibuli
fj—fossa jugulare
fc—fenestra cochleæ
fs—foramen singulare (solitarius)
f sub—fossa subarcuata
fv—fenestra vestibularis

h—hypotympanum
horiz—horizontal

i—incus

ls—ligamentum spirale

m—modiolus
me—mesenchyme
mc—membranous canal
ms—musculus stapedius
mts—membrana tympani secundaria

nc—nervus cochlearis
nf—nervus facialis
nv—nervus vestibuli

p—promontorium
pa—parantral cells
pai—porus acusticus internus
ps—perilymphatic space
psc—posterior stapedial crus
pt—perilymphatic tissue

rc—ramus cochlearis
ru—recessus utriculi
rp—ramus posterior vestibularis

sa—sacculus
sct—scar tissue
se—saccus endolymphaticus
sit—sinus tympani
s—stapes
st—scala tympani
sv—scala vestibuli
str—sinus transversus
tmp—thickened mucoperiosteum
u—utricle
vert—vertical

REFERENCES.

Alexander, G. 1922: Zur Kenntnis der Anatomie der Varietäten der physiologischen und klinischen Bedeutung des perilymphatischen Gewebes. Ztschr. f. Hals-, Nasen- u. Ohrenh., 3, p. 167-177.

1902: Ueber atypische Gewebsformation im inneren Ohr. Arch. f. Ohrenh., 55, pp. 54-58.

Altman, F. 1932: Ueber zwei seltene Anomalien der Arteria media. Ztschr. f. Anat. u. Entwickel., 97, pt. 4, pp. 509-514.

Anson, B. J., and Wilson, J. G. 1929: The Utriculo-endolymphatic Valve. Anat. Rec., 43, pp. 145-154.

Augier, M. 1930: Cartilage et osselet suprategminal sur la base endocranienne de l'homme. Paris.

Ball, James Moores. 1910: Andreal Vesalius, the Reformer of Anatomy. St. Louis.

Boisliniere: St. Louis Med. Rec., October 20, 1906.

Bast, T. H. 1929: Ossification of the Otic Capsule in Human Fetuses. Contrib. to Emb., 121 Washington, D. C.

1928: Utriculo-endolymphatic Valve. Anat. Rec., 40, pp. 665-670.

1932: Development of the Otic Capsule. Arch. of Otolaryng., 16, pp. 19-38.

Benjamins, C. E. 1913: Beitrag zur Kenntnis des häutigen Labyrinths. Ueber eine vierte Crista acustica. Ztschr. f. Ohrenh., 68.

Brock, W. 1922: Drei neue Fälle von persistierender Stapedia beim Menschen. Monatschr. f. Ohrenh., 56.

Browman, Ivar. 1899: Die Entwickelungsgeschichte der Gehörknöcheln beim Menschen. Anat. Hefte, 50, pp. 510-666.

Cirto, Alphonse. 1851: Recherches sur l'organe de l'ouie des mammifères. Ztschr. f. wiss. Zool., 3, pp. 109-169.

Cowdry, E. V. 1932: The Skin. Special Cytology, I, New York.

Crowe, S. J., and Polvogt, L. M. 1930. Embryonic Tissue in the Middle Ear and Mastoid. Arch. of Otolaryng., 12, pp. 151-161.

Cummins, H. 1925: The Vestibular Labyrinth of the Albino Rat. Jour. Comp. Neur., 38, pp. 399-459.

Dempster, W. T. 1930: Endolymphatic Organ. Jour. of Morph. and Physiol., 50, pp. 71-126.

de Brulet, H. M. 1930: Die Stellung der Maculae acusticae im Schädel des Menschen und einiger Saugetiere. Gegenbauer's Morphol., Jahrb., 65, pp. 377-393.

du Verney, Guichard Joseph. Anno MDCLXXXIV: Tractatus de Organo Auditus. Norimberg.

Eckert-Möbius. 1926: Handbuch der spec. path. Anat. u. Hist. XII, Berlin.

Eustachius. MDCCXXII: Tabulae Anatomicae, Lancisi, Amstellaedami; Albinus, CICICCCLXI, Leidae.

Fischer, J. 1922: Zur Entwickelung der atypischen Gewebsformationen im häutigen Innenohr. Ztschr. f. Mals-, Nasen u. Ohrenh., 3, pp. 177-185.

1930: Eine bisher nicht beobachtete Missbildung im häutigen Labyrinth. Ztschr. f. Hals-, Nasen- u. Ohrenh., 27, pp. 582-585.

Froriep, A. 1885: Ueber Anlagen vom Sinnesorgan am Facialis, Glosso-pharyngeus und Vagus: Ueber die genetische Stellung des Vagus zum Hypoglossus, etc. Arch. f. Anat. u. Entwickel. Leipzig.

Gage, Susanna. 1907: The Method of Making Models from Sheets of Blotting Paper. Ant. Rec., 1, pp. 166 to 169.

Guyer, M. F. 1924: Animal Micrology. p. 157. Chicago.

Guggenheim, L., and Wolff, D. 1932: Bilateral Diffuse Suppurative Labyrinthitis, etc. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY.

Guild, S. R. 1927: The Circulation of the Endolymph. Amer. Jour. Anat., 39, pp. 57-81.

——— 1932: Correlations of Histologic Observations. Acta Otolaryng., 17, pp. 207-245.

——— and Crowe, S. J., and Bunch, C. C.: Correlations of Differences in the Density of Innervation of the Organ of Corti with Differences in Acuity of Hearing, etc. Acta Otolaryng., 15, pp. 269-308.

Hammar, J. A. 1902: Studien über die Entwickelung des Vorderarms und einiger angrenzenden Organen. Arch. f. Mikr. Anat., 59, p. 413.

Henke and Lubarsch. 1926: Handbuch der spez. path. Anat. and Hist., XII, Berlin.

Hrdlicka, Ales. 1933: Seven Prehistoric American Skulls with Com-plete Absence of External Auditory Meatus. Amer. Jour. Phys. Anthrop., 17, pp. 355-379.

Karbowski, B. 1930: Vergleichende anatomische Studien über den Aqueductus cochleæ und über seine Beziehungen zum Subarachnoidalraum des Gehirns. Monatschr. f. Ohrenh., 64, pp. 687-715.

Keibel and Hall. 1912: Manual of Human Embryology, Ed. 2. Phila-delphia.

Krause, R. 1906: Entwickelungsgeschichte des Gehörorgans. Handbuch der vergl. u. exper. Entwickelungslehre der Wirbeltiere von Oscar Hert-wig. Jena, Vol. 2, chap. 6.

Lorente de No, R. 1926: Etudes sur l'anatomie et la physiologie du labyrinthe de orielle et du VIII nerf. Travaux du Laboratoire de Recher-ches Biologique de l'Universite de Madrid, S. Ramon Cajal, Vol. 24, pt. 1.

——— 1933. Anatomy of the Eighth Nerve. The Laryngoscope, Vol. 43.

Marx, Hermann. 1926: Missbildungen des Ohres. Henke und Lubarsch, ibid.

Möller, J. 1930: Das Körnersche Septum im Warzenfortsatz. Ztschr. f. Hals-, Nasen- u. Ohrenh., 26.

Meurman, Y. 1928: Zur Anatomie und Physiologie des Aqueductus Cochleæ. Ztschr. f. Laryn., Rhin. und ihre Grenzgebiete, 17, pp. 401-409.

Portmann, Georges. 1927: Recherches sur succulus endolymphaticus. Acta Otolaryng., 11, pp. 110-137.

Politzer, Adam. 1907: Geschichte der Ohrenheilkunde I, II. Stuttgart.

Retzius, Gustaf. 1881: Das Gehörorgan der Wirbeltiere. Vol. 2, Stock-holm, Köngel.

Ruttin, E. 1922: Anatomie des Utriculus und der Cysterna perilymphat-ica. Acta Otolaryng., 3, pp. 289-301.

Sakai, K. 1922 Findet sich die Macula neglecta auch beim Menschen? Ztschr. f. Ohrenh., 82, pp. 50 to 63.

Schwarz, M. 1931: Untersuchungen zur individuellen Histologie des Bindegewebes. Arch. f. Ohrenh., usw. Heilkunde, 129, Vol. 1, pp. 1-29.

Shambaugh, G. E. 1932: Cytology of the Internal Ear. Special Cytology, III, pp. 1333 ff. E. V. Cowdry, New York.

Siebenmann, F. 1905: Bardeleben's Handbuch der Anatomie des Menschen, pp. 244 ff. Jena.

1922: Ein neues Labyrinthmodell des menschlichen Gehörorgans. Zeitschr. f. Ohrenh., 82, pp. 1-4.

Singer, L. 1932: Ueber entzündliche Erkrankungen des Mittelöhres und der pneumatischen Hohlräume des Schläfenbeines. Ztschr. f. Ohrenh., 32, p. 130.

Streeter, G. 1918: The Histogenesis and Growth of the Otic Capsule and Its Contained Periotic Tissue-spaces in the Human Embryo. Contr. to Emb. No. 20, pp. 1-54, Washington, D. C.

1916: Über sogenannte atypische Epithelformationen im häutigen Labyrinth. Eine rudimentäre Macula neglecta. Morphol. Jahrbuch, 44, p. 403 ff.

Tandler, J. 1902: Zur Entwickelungsgeschichte der der Kopfarterien bei den Mammalia. Gegenbauer's Morphol. Jahrbuch, 30, p. 275.

Terry, R. J. 1917: The Primordial Cranium of the Cat. Jour. of Morphol., 29, pp. 281-434.

1919: The Relation of the Facial Nerve and Otic Capsule. Amer. Anat. Rec., 17, pp. 235-242.

Toynbee, J. 1853: Diseases of the Ear. Trans. Path. Soc. of London, 4, pp. 251-260.

Valsalva. 1707: De Aure Humana Tractatus. Trajecti ad Rhenum. Ex Officina Guilielmi vande Water Academiea. Typographic I, i ccvii.

Vesalius, A. MDCCXXV: Anatomica and Chirurgica. Lugduni Batavorum.

Voltolini. 1860: Anatomische und pathologische-anatomische Untersuchungen des Gehörorgans. Virchow's Arch. f. path. Anat., 18, pp. 34-50.

Weber, Moritz: 1930: The Bone Picture of Otosclerosis. Arch. of Otolaryng., 12, pp. 608-686.

Werner, C. F. 1932: Ueber Wachstumsprocesse in der normalen Labyrinthkapsel und ihre Beziehungen zur Otosclerose. Arch. f. Ohren-, Nasen- usw. Heilkunde, 129, pp. 128-149.

Wilder, Alexander. 1901: History of Medicine. New Sharon, Maine.

Wittmaack, K. 1926: Die Facialislähmung. Handbuch der spez. path. Anat. u. Hist., XII, Henke and Lubarsch, Berlin.

1911: Ueber sogenannte atypische Epithelformationen im membranösen Labyrinth. Verh. deutsch. otol. Ges., 20, pp. 406-408.

Wolff, D. 1931: Melanin in the Inner Ear. Arch. of Otolaryng., 14, pp. 195-211.

Zuckerkandl, E. 1873: Ueber die Arterie Stapedia des Menschen. Monatschr. f. Ohrenh., Jahrgang VII, p. 5, Berlin.

XVI.

PRIMARY ACTINOMYCOSIS OF THE NOSE WITH EXTENSION TO PHARYNX, HARD AND SOFT PALATE, AND CERVICAL VERTEBRÆ, WITH REPORT OF CASE.

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Actinomycosis was first recognized as an infection in cattle by Bollinger[1] in 1877, and one year later described by Israel[2] for man. Several years later Ponfick[3] recognized the identity of the disease in man and animals.

Although actinomycosis occurs most frequently among cattle and humans, it is also found in sheep, pigs, cats and horses. In humans, age apparently bears no relation to its activity. New and Figi[8] report 157 cases, the youngest a patient 9 years of age, and the oldest, 70. Stokes[10] reports a case in an infant, 4 weeks old. Males are more frequently affected than females. While the disease is most frequently found in farmers and their co-workers, it cannot be said to be due to occupation, since in the literature we find actinomycosis reported also in an office girl, a meat cutter, a physician, a stone mason, a judge, a chiropractor, a barber, a miner, a cook, and those representing many other occupations.

In cattle, the disease usually shows itself as a swelling of the jaw with sinuses and purulent discharge, in which peculiar granules or sulphur grains are found. In humans, the affection is also quite commonly a swelling of the jaw, but typical lesions are occasionally found in the thoracic organs, intestines and skin.

The organism itself grows in tangled mycelia, and is characterized by the sulphur grains which are knots of mycelium with radially projecting tips which form a layer covering the central tangle. Each tip is surrounded by club-shaped or bulbous covering of refractive material. Microscopically, sections through such a granule show a scalloped margin of such clubs arranged radially. The granules average about 0.5 mm. in diameter and the

actinomyces are approximately the thickness of an anthrax ba-
cillus, and, according to Babes,[4] contain granules of varying size.

Bostroem[5] cultured the organisms aerobically, but Wright,[6]
Israel and Wolf[7] all agree that the organisms grow best under
anaerobic conditions. Animal inoculations carried out with both
pus and pure cultures, have given very little results. Transmis-
sions from animal to animal, or animal to man, have not been
proven.

It is generally thought that the most common means of infec-
tion is either direct contagion from lower animals or through the
agency of hay, straw or grain. Berestnew[9] was able to isolate
actinomycosis from straw, grass and hay. New and Figi doubt
direct contagion, but believe the presence of the disease indicates
abundant organisms on the vegetation of the locality. By many,
it is thought the infection enters the tissues through chewing
splinters, straw, grass, etc. Others deny this possibility and state
that such practices furnish only the trauma, by which organisms,
normally present in tonsil crypts and carious teeth, may display
their pathogenic properties. (Wright and Lord.[11])

Once the actinomycosis has reached the tissue, the cells are
quickly necrosed, aided by an abundant accumulation of leuco-
cytes. The process advances slowly, and there is a profuse
formation of granulation tissue around and about such an area
after a few weeks or months, while the central portion of the
lesion is made up of liquid pus, full of branching organisms. The
outer zones are composed of such dense, fibrous tissue, as to
form a tumor-like mass. Lining the cavity are still fresher gran-
ulations, loaded with large mononuclear, wandering phagocytes.
At times the encapsulated pus may become calcified and sponta-
neous cure follow; this, however, is the exception, since the
diseaes is usually one of the greatest chronicity. The mycelium
grows and advances into the tissues slowly, completely destroy-
ing everything it comes in contact with, and stopping at nothing;
bones are penetrated as easily as muscles. The process is attended
by a tremendous formation of scar tissue.

Diagnosis is made by finding the ray-fungus or actinomycosis
under the mircroscope in sections of tissue removed from the in-

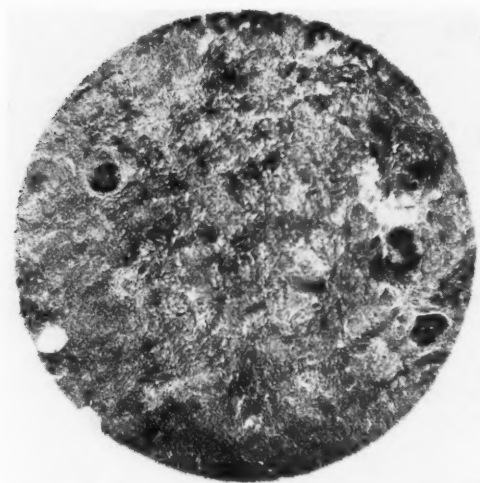Fig. 1. External deformity of the nose with prolific scar formation, due to actinomycosis.

Fig. 2. Three colonies of actinomyces in a section of tissue removed from the pharynx of patient, whose case is reported in this paper.
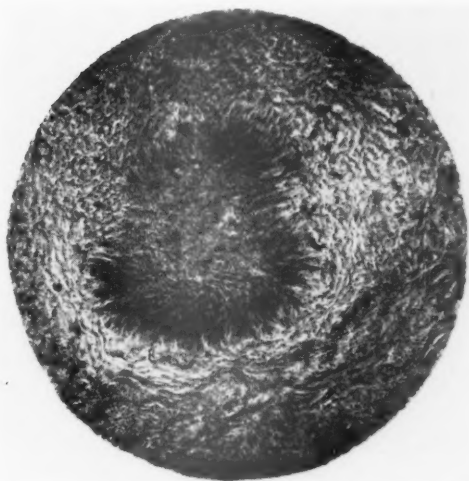
Fig. 3. One colony of actinomycosis under oil immersion from the same patient. Note dense, fibrous tissue surrounding periphery.
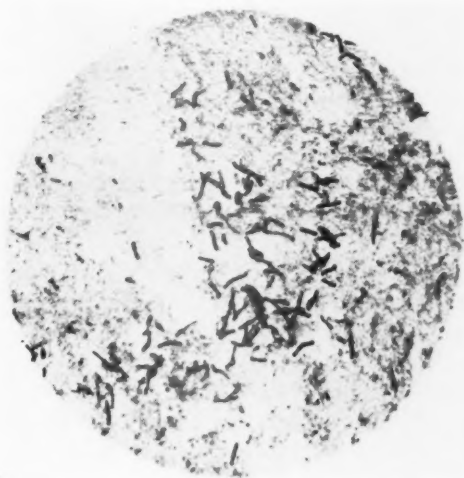
Fig. 4. Actinomyces from the same patient.

fected area. Actinomycosis must be differentiated from tertiary syphilis, tuberculosis, carcinoma, sarcoma, fibromata, cellulitis, coccidioides—granuloma and rhinoscleroma.

The treatment is surgical where possible. Palliative treatment consists of general constitutional measures, including iron injections to combat anemia which is usually present. Montgomery[12] reports a case in a girl, 12 years of age, treated locally with copper sulphate crystals and internally with large doses of potassium iodid, who recovered. Vaccines, both autogenous and stock, proved of little value. Radium has been tried, but results have been disappointing. X-ray in large doses seemed to give fair results. Eiken[23] gives ½ skin erythema dose, .5 mm. Al. Six treatments in the course of nine months. Goettsch[22] gives 25 per cent erythema dose .5 mm. Cu.; 180 Kv. for several treatments in facial and neck actinomycosis.

Dr. Jackson-Coates' book ("The Nose, Throat and Ear")[24] states that actinomycosis is rather a common cause of granulomata of nose and sinuses. I have been unable to find any specific case reported as having its primary lesion in the nose. Magnotti[13] reports two cases of actinomycosis of the nasopharyngeal tonsil. Vermes[14] reported a rare complication following tonsillectomy performed because of recurrent angina. A deep pharyngeal abscess developed which showed actinomycosis. Hofhauser[15][16] reports a case of peritonsillar infiltration and abscess. The same author also reports the presence of actinomycosis-like bodies in the palatal tonsils, but states they are distinguishable microscopically, and suggests they should be called "corpora lacunaria," to avoid confusion. Wilkinson,[17] Wright,[18] Castellani,[19] Bell,[20], Grosvenor,[21] all report similar actinomycosis-like granules in normal tonsil crypts, but feel that these organisms are probably nonpathogenic. New reports two unusual nasopharyngeal tumors, the first tumor is that of actinomycosis of the nasopharynx, located in the vault on the left side and almost protruding to the midline. Radium treatment was given, seven weeks afterwards a phlegmon ruptured in the left temporal region, just above the zygoma, from which actinomyces were demonstrated grossly and microscopically.

Report of Case: M. C., Mexican woman, age 54. Entered hospital January 13, 1932.

Complaint.—One month ago, patient had a nasal plastic operation performed. She states that at the time of operation, her throat was injured in two places. One wound healed rapidly, but the other became infected; ulceration and swelling followed. At the present time, she has difficulty in swallowing, and is unable to speak clearly.

Family History.—One brother died of scarlet fever at three years of age, and one sister died of smallpox at two years of age. No family history of cancer, diabetes, tuberculosis, renal or mental diseases.

Past History.—Born on a cattle ranch at Sonora, Calif., in 1878. At the age of ten, while playing, she scratched her face on the limb of a tree—the scratch extended across her cheek, into her left nostril. The wound seemed of no consequence at the time, but shortly afterward a "very bad infection" started in her nose, with the formation of "much pus." This was followed by a marked deformity of the nose—the infection spread to the skin around her mouth and finally ate a hole through her palate into her mouth. Her tonsils subsequently became infected, and she developed large glands of the neck, which were diagnosed, mumps. She remembers that many of the cattle on the ranch where she lived had lumpy-jaw.

The mumps ulcerated and expelled pus through the skin. She was taken to the doctor who prescribed some pills, and blue colored ointment, which was rubbed into the lumps of her neck and into her thighs every night for several weeks. At this time she was acutely ill, but recovered after several months. Since that time she has suffered periodically from sore throat and swellings in her neck. In 1904 while working in a hospital, she was given a series of treatments of medicine injected into the arm, and rubs with blue ointment. In 1918 the patient entered Lane Hospital for hysterectomy. Her history at that time describes her nose and throat, but makes no mention of any glandular enlargements. Her Wassermann test was negative, and her recovery uneventful.

In 1922 she was admitted to a hospital, where she gave the following history: For five years she had noticed a lump about the size of the end of her thumb on the left side of her neck, at the angle of the jaw—more noticeable when chewing food. This lump became painful on March 18th. By March 20th she was unable to swallow, and had great difficulty breathing, because of the swelling.

Examination at this time showed the patient unable to open her mouth more than half an inch, because of the large swelling on the left side of the floor of her mouth. The whole left side of the neck was swollen and tender, but was not covered with reddened skin. Diagnosis was Ludwig's angina. An incision was made into the neck externally, about three-fourths inch below the ramus on the left side—no pus was found. A hemostat was forced into the swelling under the tongue in the region of the opening of the sublingual duct—10 cc. of thick whitish pus was evacuated. The patient was dismissed March 30th, much improved.

Four nasal plastic operations were performed between December, 1930, and January, 1932. The patient stated that during her early life she had many times been diagnosed as syphilitic and treated for lues, but at no time was her Wassermann positive.

Physical Examination.—On entering the hospital January 13, 1932, examination showed a marked deformity of the nose externally. Both nares were practically closed by scar tissue. A rhinoscopic examination showed a great loss of tissue with the anterior two-thirds of the septum missing, and the turbinates indistinguishable. The nasal cavity was filled with crusts and foul smelling discharge.

Throat.—Showed a greatly scarred and contracted palatine arch, with absence of the uvula. Arising from the upper left pharynx was a large white sloughing mass which extended downward into the hypopharynx, and obstructed the view of the larynx. A tentative diagnosis of carcinoma or lues, was made. A biopsy was taken which showed deep staining bodies which presented the typical ray-fungus appearance. Myceleal bodies were also found throughout the sections. On February 15th, after repeated removal of the slough, the posterior pharyngeal wall showed a large mass of pouting, granulation tissue, which exuded a creamy white pus; no sulphur granules were to be found. The neck stiffness and pain was so severe by this time, that she could no longer move unassisted. On February 24th a small spicule of bone was seen free in the mass and removed. From this time on, bone could always be palpated on the posterior pharyngeal wall. Her condition gradually became worse until March 18th, when her right arm and leg became paralyzed, and she died rather suddenly.

Clinical diagnosis was:

1. Terminal bronchopneumonia.
2. Actinomycosis of pharynx with osteomyelitis of the first and second cervical vertebrae with meningitis.

While in the hospital she received massive doses of X-ray and K. I. to tolerance. At autopsy the anatomic diagnoses were:

1. Strepthothricosis of pharynx, with erosion of cervical vertebrae, and limited meningitis of spinal cord.
2. Chronic glossitis.
3. Chronic laryngitis.
4. Osteomyelitis of first and second cervical vertebrae.
5. Bronchopneumonia.
6. Meningitis.

SUMMARY.

1. Primary lesions of actinomycosis may occur in the nose.

2. The disease may be quiescent for years and then reappear. The organisms invade bone as easily as muscle.

3. The destruction of tissue by actinomycosis is always followed by great formation of encapsulating fibrous tissue—this makes the disease one of great chronicity.

4. The lesions of actinomycosis may be easily mistaken for tertiary syphilis.

FITZHUGH BUILDING.

REFERENCES.

MacCallum, W. G.: Text Book of Pathology. W. B. Saunders, 1921.

Hiss & Zinsser: Text Book of Bacteriology. D. Appleton, 1914.

1. Bollinger: Deutsch. Zeit. f. Thiermed. Ill., 1877.

2. Israel: Virch. Arch., 74, 1878 and 1879.

3. Ponfick: Die Actinomykose d. Menschen, Berlin, 1882.

4. Babes: Virch. Arch., 105, 1886.

5. Bostroem, O.: Beits. Z. Path. Anat. u. z. Allg. Path., IX, 1890.

6. Wright, J. H.: Jour. Med. Res., VIII, 1905; 349.

7. Wolf and Israel: Virch. Arch., 126, 1891.

8. New, G. B., and Figi, F. A.: Actinomycosis of Head and Neck. Surg. Gyn. & Obst., 37:617, 1923.

9. Beresthew: Centralbl. f. Bakteriol., 24, 1898.

10. Stokes, W. R.: A Study of Group Actinomycosis in the United States. Arch. of Surg., 11:809 (December), 1925.

11. Lord, F. T.: A Contribution to the Etiology of Actinomycosis. Boston M. & S. J., 163-82 (July), 1910. Etiology of Actinomycosis, J. A. M., 55:1261, October 8, 1910.

12. Montgomery, G. W.: Actinomycosis of the Nasopharynx and Soft Palate. N. W. Med., 24:344, 1925.

13. Magnotti, T.: Two Cases of Actinomycosis of the Nasopharyngeal Tonsil. Arch. ital. di otol., rinol. e laringol., 39:96, 1928.

14. Vermes, E.: A Rare Complication (Actinomycosis) After Tonsillectomy. Wien. med. Wchnschr., 2:1602, 1931.

15. Hofhauser, J.: Tonsillar and Peritonsillar Actinomycosis. Arch. f. Ohren-Nasen u. Kehlkopfh., 124:44, 1929.

16. Hofhauser, J.: The Presence of Actinomyces-like Bodies in the Palatal Tonsils. Arch. f. Ohren-Nasen u. Kehlkopfh., 129:293, 1931.

17. Wilkinson, H. F.: Pathologic Changes in Tonsils. Arch. Otol., 10:127, 1929.

18. Wright, J.: Actinomycosis of the Tonsils. Am. J. Med. Sci., 128:74, 1904.

19. Castellani, A.: Douglas, M., and Thomson, T.: Further Observations of Tonsillomycoses. J. Trop. Med., 26:19, 1923.

20. Bell, H. H.: Bacterial Flora on the Faucial Tonsils. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 33:305, 1924.

21. Grosvenor, L. N.: Actinomyces in Tonsillar Crypts. Ill. Med. J., 19:513, 1911.

22. Goettsch, H. B.: Roentgenotherapy of Actinomycosis. Nederl. Tydschr. Geneesk., 2:4645, 1930.

23. Eiken, T.: Diagnosis and Treatment of Actinomycosis. Ugeskr. Laeg., 2:581, 1929.

24. Spencer, Frank R.; Jackson, Coates: The Nose, Throat and Ear, 1:187.

XVII.

THE NEED OF A REVISED NOMENCLATURE OF CHRONIC PROGRESSIVE DEAFNESS.[*]

Thomas J. Harris, M. D.,

New York.

At the Atlantic City meeting of this society in 1929, a paper was read by Dr. James G. Dwyer on "The Classifications of Deafness." The paper and the discussion which followed set forth the obsoleteness of the present nomenclature and the need of a new one in keeping with present day views. In the time which has elapsed since this meeting, the National Conference on Nomenclature of Diseases has been actively at work in the preparation of a new nomenclature covering the entire field of medicine and has compiled the result of its labors in an elaborate volume intended for general use. This society was a constituent member of the conference and was represented in its deliberations. The Council gave serious consideration to the proposed nomenclature but did not feel it was prepared to recommend it to the society. Modern otology is of comparatively brief duration. Indeed it is covered by the life of this society, and those who were responsible for its development were known to many of its present members. The nomenclature in use today dates from the last thirty years of the last century. It is that which is found in all the standard textbooks of that period such as those of Bezold, Gruber, Roosa and Politzer.

Among all of these, that of Politzer[1] sets forth the grounds for such classification most clearly. It was his opinion that a classification of middle ear disease, either on an etiologic or patho-anatomic basis, is impossible. He goes further in stating that he did not agree with the contention that the various inflammatory processes in the middle ear are only different steps of the same disease. Based on his wide experience, he believed that while it

is possible for one form to transform into another, certain definite characteristics distinguish different forms of middle ear disease from beginning to end. In view of this, Politzer maintained that the clinical basis is the only one for classification. In common with all other otologists of the period, he divided nonsuppurative middle ear diseases into two distinct classes. To the first he gave the name of "*middle ear catarrh*," a condition which he recognized as involving both the middle ear and eustachian tube, which is distinguished by "a swelling of the mucous membrane with a throwing out of serous or mucous secretion." This condition can entirely subside or there can take place the formation of connective tissue bands with resultant alteration in the position of the drum membrane and ossicles.

Closely related to this first group of cases, but in many instances without any preceding secretion, is the second large group which he called "*dry catarrh*" of the middle ear, an insidious process resulting in the formation of adhesions and the binding down of one or more of the ossicles. Politzer continued to teach this classification until he had completed his classical work on the labyrinthine capsule. As a result of this and of the work of others he adopted, as set forth in the 1908 edition of his textbook, in place of the term "dry catarrh" or "adhesive catarrh" to designate the second group of middle ear diseases, the name in use today, "*otosclerosis.*"

In Politzer's opinion, otosclerosis is a primary affection of the labyrinthine capsule and (ordinarily) the middle ear is not involved, although occasionally there is a mixed affection of middle ear catarrh and otosclerosis. This abandonment by Politzer of the term "adhesive catarrh" has not been followed by all writers on the subject. Jacobson and Blau[a] cling to the two forms of moist and dry catarrh and dispute Politzer's revised classification of chronic adhesive process of the middle ear and otosclerosis, on the ground that they are both alike in symptoms after a time and also because, as he admits, the two can be mixed. To quote Politzer: "In a number of cases in which there are pathologic changes in the membrana tympani and a slight impermeability of the tube, the progressive course, the subjective symptoms and the results of the hearing tests are the same as in typical

cases of otosclerosis. In many it is exceedingly difficult to make a differential diagnosis. This is based on the fact that, besides adhesive changes in the tympanic cavity, otosclerosis changes are also found in the labyrinthine capsule. Such mixed forms occur rather frequently. It is impossible to make a positive diagnosis, because the catarrhal changes localized in the middle ear often present the same clinical picture."

Phillips in the sixth edition of the "Diseases of the Ear, Nose and Throat," 1922, divides diseases of the middle ear into those which are nonbacterial and those which are bacterial in origin. The nonbacterial diseases of the middle ear, he states, are known as "catarrhal," and those of bacterial origin as "inflammation." The catarrhal type is due to mechanical effects produced by closure of the eustachian tube, while those of bacterial origin are the result of the invasion of micro-organisms. This classification shows how much Phillips is at variance with the modern views of bacteria being the cause of disease of the ear. Kerrison, in the latest edition of his textbook on the ear (1930), follows the classical division of (a) otitis media catarrhalis chronica, (b) otitis media catarrhalis hypertrophica, and (c) otosclerosis.

Logan Turner, in his "Diseases of the Nose, Throat and Ear" (1924), divides cases of deafness into "those caused by past attacks of catarrh or suppuration in middle ear cleft—i. e., chronic adhesive process in the middle ear or cicatrized tympanum, and those due to otosclerosis," while Keeler, in his textbook on "Modern Otology" (1930), recognizes two general classes, first, the hypertrophic, often called the "exudative" or adhesive catarrh, and second the atrophic form, known as "dry" or "sclerotic," under which otosclerosis was formerly classed. In other words, Keeler still adheres to the time honored classification which existed before the recognition of otosclerosis.

These quotations from present day writers would seem to convey the idea that the nomenclature so long in use is entirely satisfactory. The position, however, taken by Dwyer and by those who took part in the discussion of his paper four years ago, is strongly opposed to this. The present classification, in his opinion, is a "waste basket for numerous unknown conditions. Pathologically the term 'catarrhal' is incorrect. The present classification

does not cover etiologically those conditions that have been rec-
ognized in the last few years, such as focal infection and that
of the endocrin type." He urges that the term "catarrhal" should
be dropped because it does not describe the condition that is pres-
ent but psychologically gives a false impression. In the discussion
which followed, Duel also urged that the term "catarrhal" should
be dropped. He favors the term "hyperplastic," acute or chronic;
or atrophic, acute or chronic, and dwells on the importance of the
diagnosis of toxic infection of the eighth nerve, while Emerson
is of the opinion that a *catarrhal process* is a mild inflammation
compared with more virulent attacks involving the middle ear.
In all cases, in his opinion, the cause is a toxemia proceeding
most usually from the tonsils. In other words, in every infection
that involves the middle ear, whether it is a catarrhal process or
a more virulent attack, there is at the same time damage to the
perception apparatus which is intermittent and in which the loss
of tone perception takes place in an orderly fashion, until we
finally get lowered bone conduction in all. The changes in the mid-
dle ear are coincident with the damage to the perception apparatus
and cannot be separated from it. In line with Dwyer's paper and
the discussion which followed are the views expressed by Ko-
petzky in his paper on the "Nature of Progressive Deafness,"[2]
who believes that (1) "Progressive deafness is not a distinct
entity; (2) in most instances progressive deafness is a local
manifestation of a general metabolic disorder, and (3) that the
present classification of deafness is a hindrance to investigation
and research in that it offers both a hypothesis and a conclusion."
It is Kopetzky's opinion that the entire nomenclature of deafness
requires revision, since it is based on results obtained by a technic
of testing the hearing which at best is only an approximation.
These views of Dwyer, Duel, Emerson and Kopetzky show that
there is far from unanimity among those whose opinions are to
be valued in regard to maintaining the present nomenclature.

There appears to be an increasing sentiment that the term "ca-
tarrhal" is falsely used. The dictionary defines "catarrh" to be an
"inflammatory affection of a mucous membrane, especially of the
nose or air passage." This would indicate that the objection to
its use in connection with inflammatory disease of the middle ear

is not well founded. The most that can be said is that its employ-
ment is unnecessary and on that account it could, without loss,
properly be discarded. It has been already stated that Politzer
was opposed to any classification on a pathologic or etiologic
basis. This opinion, coming from so great an authority, cannot
lightly be dismissed. More and more, however, the importance
of taking into consideration pathology and etiology is being
stressed. The new national nomenclature is based upon etiology
plus anatomic location. If a change is to be made in the present
nomenclature, the new one must be one that can be used not
merely in the office and the hospital but in the clinic, where the
large number of patients seen does not at the time permit an
extensive investigation to determine the entering diagnosis. In
recent years the term *progressive deafness* has come into use with-
out any accepted definition. Its employment interchangeably with
the term "otosclerosis" manifestly is wrong. It should be used
to cover all cases of advancing deafness and so would include
chronic otitis media, eighth nerve toxemia and otosclerosis.

The term "otosclerosis," regarded by many as a misnomer, has
only served to increase the difficulty of effecting a satisfactory
revision of the nomenclature. As has been pointed out, there
exists a decided difference of opinion whether otosclerosis at
times has its origin in the middle ear or whether we are really
dealing in certain cases with a mixed form of disease, as taught
by Politzer. Metzianu,[5] on the basis of his clinical studies, claims
that otosclerosis begins with a catarrh in the eustachian tube and
the middle ear. According to Gray,[6] such changes can be present
but are not essential. Habermann, in postmortem findings in a
case of otosclerosis, found evidences of old middle ear inflam-
mation. Steinbrugge,[7] De Lemos[8] and Denker[9] all believe that
middle ear changes exist or can exist.

What we have said makes plain the difficulty of the problems
to be solved. The society has for the past few years distinguished
itself by the important scientific contributions that have been made
before it. As members we are proud of this and would not have
these contributions lessen. We are, however, for the most part
clinicians, and problems that have to do with our every day
clinical work must remain of prime importance. Nothing can be

more important than the determination of a correct classification
of the cases of ear disease, especially of the middle ear, that
present themselves to us. The classification adopted by the
National Conference on Nomenclature may be the most satisfac-
tory one. This can be determined only by suitable investigation.
We would accordingly renew the recommendation made at the
Atlantic City meeting of four years ago that a special committee
be appointed to consider the proper nomenclature of middle ear
disease and report at a future meeting of the society, to act, if
it seems desirable, in conjunction with similar committees from
other national societies. Such a committee would determine
whether the classical view of Politzer that the classification of
chronic deafness shall be made from the clinical picture alone
must, in view of the widened knowledge of disease of the ear,
be abandoned; and etiology, as in the new nomenclature of the
National Conference on Nomenclature, or pathology be substi-
tuted. This committee also will have as its task to say whether
the form of progressive deafness commonly called otosclerosis
is an actual entity and, if so, whether involvement of the middle
ear forms part of this entity, at least in certain proportion of
cases, or whether it is only coincidental with the middle ear
process; and finally it will be the duty of the committee to desig-
nate by a proper term that large group of cases of chronic deaf-
ness with recognizable middle ear changes which we at present
call otitis media chronica catarrhalis, admitted by everyone to
be a lazy and unscientific term and one that calls for speedy
correction.

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BIBLIOGRAPHY.

1. Politizer: Lehrbuch der Ohrenheilkunde, 1893; p. 214.
2. Kopetzky: Nature of Progressive Deafness. Archives of Otolaryn-
gology, 1927; Vol. 5, p. 464.
3. Jacobson and Blau: Lehrbuch der Ohrenheilkunde, 1902; 3rd Edition.
4. Transactions of American Otological Society, 1929; p. 76.
5. Metzianu: Otosclerosis. American Otological Society; Vol. 1, p. 322.
6. Gray: Otosclerosis. American Otological Society; Vol. 1, p. 322.
7. Steinbrugge: Otosclerosis. American Otological Society; Vol. 1,
p. 307.
8. De Lemos: Otosclerosis. American Otological Society; Vol. 1, p. 309.
9. Denker: Otosclerosis. American Otological Society; Vol. 1, p. 309.

XVIII.

ECONOMICS IN OTOLARYNGOLOGY.*

Burt R. Shurly, M. D.,

Detroit.

"So fleet the works of men
Back to the earth again
Ancient and holy things
Fade like a dream."

We are gathered together in the greatest epoch-making era known to the people of this marvelous and glorious nation of ours. So rapid is this realm of readjustment, of evolution and of revolution, that it is only necessary to peruse the morning page, and our many medical journals, to realize that the old and the new in economics and medicine are caught in a whirling cauldron, churn or wheel of fortune, and the results are speculative problems that make fascinating argument, where bevies of words, opinions and thought are bantered back and forth, like the old game of battledore and shuttlecock.

It seems only yesterday when the equipment of an otolaryngologist was a galvanocautery, a few specula, and spray bottles, a Jarvis snare, a Matthieus tonsillotome, a few forceps, a bottle of rhinitis tablets and, with a smile of good cheer, you were called a specialist.

Thirty-five years of progress have obliterated the ancient ideas of medical economics—and how was this accomplished?

We are in the home of "great men, great events, and great deeds, that grow as we recede from them, and the rate at which they grow in the estimation of men is in some sort a measure of their greatness." Tried by this standard, the establishment and development of organized medicine here has done as much for the relief of human suffering and the progress of our specialty and for medical research as any one contribution of the century.

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*President's address before the American Academy of Ophthalmology and Otolaryngology. Boston, September, 1933.

One of the great professional pleasures of my life has been service on the American Board of Otolaryngology, which signal honor is most highly appreciated by me. Experience in the examination of men, more than 2,000, so enthusiastic and many so well prepared for their life work, is an education and delight linked with labor and responsibility. An experience of this sort gives me an insight into numerous factors that are exerting a tremendous influence in the trend of our specialties.

In a careful analysis of these examinations one instinctively asks, Are they superficial, insincere, or simply a matter of form? Those of us who have spent many years in teaching realize the difficulties of the student and the teacher. We know that examinations do not necessarily test the knowledge as we might wish. It is impossible that the written or oral examination in the short space of time allotted will give complete evidence of the various qualities essential to safe practice. We believe, however, that this time-honored custom, conducted with dignity, sincerity and, above all, with justice and common sense, is our best method to establish high standards of safety, to stimulate study and to realize the importance of never-ending responsibility in the care of the sick.

Fairness and honesty of purpose in these examinations deserve your whole-hearted support and co-operation. I look to the day in the near future when every hospital and medical school will require certification before a staff appointment in the senior or junior attending or teaching service may be held. Postgraduate and all medical education, which is inseparable from better medical economics, is keeping pace with the demands of higher standards, and it is no longer necessary to seek foreign fields of instruction when the job is well done at home in many of our great medical centers. These Boards need your helpful and earnest co-operation to the end that we may lead the way to a confederation of our special Boards that will include an Advisory Council of at least twenty of the specialties that are striving for higher standards and better postgraduate instruction.

The advancement of opportunity, in part, rests in the study of medical research, while this Society recognizes in the liberal use of its funds. The progress of our specialty requires a great cen-

tralized library, which we are attempting to establish at Washington, with the splendid co-operation of our Surgeon-General of the Army.

The day of marbled halls for hospitals and colleges and schools is rapidly fading to a simple, substantial architecture that economizes for the practical good and usefulness of that which may be found in the interior.

Our new economic era demands a changing psychology, a prompt readjustment of medical costs, sharp lines of definite form must work out rapidly as to where state medicine leaves off and the private practitioner of the specialty begins. The clinical assistant must be paid that he may live to learn, and our private hospitals must be subsidized with state and municipal funds that they may carry on a system of financial remuneration for the staff

Those of us who served in the World War well remember that there were no specialists in the Army and Navy prior to this great event. It may be worthy of history to record again that Dr. H. P. Mosher, Dr. Charles W. Richardson and your speaker were a committee of three who met with the Surgeons-General in Washington and established Oto-Layrngology as the first special branch in the service. This was quickly followed by Drs. Parker, Black and Boardley, in Ophthalmology.

The strenuous work and worry of those days could be prevented in the future if our younger specialists would join the Medical Reserve Corps and in time of peace prepare for war, as Washington said.

With an unswerving optimism and enduring appreciation we look to the younger otolaryngologist to balance his wonderful training as a skilled technician with the older school of conservatism and bedside psychology, and realize that judgment, experience, and the art of otolaryngology are estimable qualities, attained by long years of toil and mistakes in the operating room and at the bedside.

What is more fascinating and useful and practical than the application of the grand special education of our membership to the problems of the school child, where changing economics is the order of the day? If we are valuable to our people now, that value will increase an hundredfold if we become members of a

Board of Education or examining specialists for a school system that must meet our problems in their incipiency. The periodic or insurance examination comes too late for early prevention and diagnosis. We must solve the problem of our children in the kindergarten and the first grades.

The World War brought out, during enlistment, a deplorable and astounding number of physical defects among our young men. A great additional work could be done if every Board of Education had a certified ophthalmologist and otolaryngologist on the staff and that these men organize throughout the country for the discussion of defects within our respective fields.

Our own school system in Detroit has developed along these lines extensively, and an excellent School for the Deaf, Hard of Hearing and sight-saving classes are carefully conducted with the supervision of specialists.

If we are to successfully combat the incipient weaknesses and physical defects of the race, and save millions of dollars in public welfare expense, I believe that the generalship, supervision, planning and special study and research would fall in a great measure within our special domain. Tonsils and adenoids, deafness, sinus infection and attending foci, with a lack of resistance, come within our field in their incipiency, and remedies and procedures, the economic problems, the research studies and the direction and supervision should be led by the otolaryngologist if prevention of future trouble can be attained.

The present era is given to words that flow like the babbling brook. If any I may utter could echo and re-echo down through the corridors of time, I trust that the medical trail of the future may be emblazoned with our specialties that recognize the sterling worth and value of medical research.

That a corps of young men be developed and maintained under governmental, university or allied special society guidance, who will select and direct problems of research within our field. A successful co-operation and collective investigation of our five national societies, with definite plans of an advisory committee, would concentrate the efforts of many laboratories and workers on a series of centralized problems.

The future of preventive ophthalmology and otolaryngology includes an enormous field of study and opportunity to benefit humanity. Our trail leads onward and forward. The dawn of a new prosperity is breaking, and science and specialism will turn our dreams to reality, our hopes to a final solution, and our hearts' desire, the good of mankind, will prevail, even to the day of judgment, when the gates will be thrown asunder and the place that knew him shall know him no more.

Can medicine stand on the Rock of Gibraltar and defend the fundamentals of the older days or must we give way more to the insurance company, and corporations, state and chain store medicine?

I do believe that the private hospital must be subsidized and the clinic and staff assistants well paid, although we are neither a trade nor an industry.

The layman seldom has the choice of a physician or surgeon—one is thrust on him. When you ask who did the operation you are told that I don't know, the blank hospital did it. The people are restless. They consult a friend's book, perhaps of seventeen specialists, a chiropractor or an osteopath, and if they sneeze they choose an allergist; a headache requires a brain surgeon; a lumbago, an orthopedist; an enlarged lymphatic gland, a gland or endocrine doctor; a cough, a tuberculosis specialist. The child requires a pediatrist up to 16 years of age; and if you are constipated you need a rectal surgeon because your rectal valves are too stiff. Loss of sight requires a consultation with the optometrist; cervical adenitis is rubbed out by the osteopath. Those injured in the eye or ear are taken to the industrial surgeon or the receiving hospital, who receive and take all, sending the old cases to the county farm. If a nickel is lodged in the throat, the emergency ward takes them in, and the bronchoscopist who is a thoracic surgeon gets the nickel. Intubation is done in the contagious hospital by the epidemiologist and the tracheotomy by the general surgeon. Cancer of the throat is treated by the cancer surgeon, and syphilis is treated by the dermatologist. The family physician takes our tonsils. We will have left to us cataracts, trachoma, cross eyes and a few foreign bodies in the eye, sinus

disease, deflected septa, running ears; and so-called catarrh will come to the otolaryngologist if we do not fight and defend ourselves, which we will.

The people have become derelicts on the ocean of a half-baked medical education furnished by us over the radio, in the office, at the evening society event, in the drug store, the newspaper, and we have been trying to educate a people that cannot understand.

In the older days a plan of treatment belonged to the doctor and his prescription was his, in Latin. Now the proprietary house dictates his treatment and his psychology with preparations that become household remedies because he has told the family all they want to know. They save money and his services are no longer needed.

It is said that doctors charge too much—quite true in some cases—but the facts are they *charge* and *do not collect* too frequently—and each new patient is not met with an understanding of medical costs. An office assistant trained to do this work has proved highly satisfactory. In this way I have no financial contact with the patient, and my study of the cases is on a professional basis only.

Why do the utility company, a monopoly, the big dry goods house, the auto factory and hundreds of industrial plants practice medicine? My only answer is that we are Russianized and socialized because our protest is too feeble. We have no power in legislative halls. Our leaders have placed the stigma of politician with a slur of disgrace and innuendo upon all who run for office. It is essential for our defense and relief from serfdom that key positions in the municipal, state, governmental and educational bodies shall be held or strongly influenced by doctors selected and appointed to office who have the welfare of organized medicine at heart.

We plead that big business give back the practice of medicine to the doctor, or that we organize a defense from our national and special societies that will present our plans of relief in person to all great executives and beg them to recognize the right and justifiable plea that all work should be referred to the family doctor and by him to the specialist. This will solve the problem of

the recovery act so far as medicine is concerned and lead the way
to the employment of more nurses, doctors and hospital beds.

If we could be satisfied with the U. S. P., and N. F., in thera-
peutics and not tell the public the name of proprietary remedies
to put in the eye, ear, nose and throat, the pharmacist and doctor
would get on better, and the patient would not run wild in
recommending to his neighbor a long list of advertised prepara-
tions that are used at random.

The "New Deal" in medicine will have the following remedies.
It will not be necessary to wear a colored buzzard on your chest
or fly the flag in the office; but the "Recovery Act" for the spe-
cialist has the slogan—back to the general practitioner, who must
be paid a just fee promptly.

A return to a normal fee schedule; no overcharge for the sup-
port of marbled hospitals. Lessened fees for diagnosis, by the
elimination of unnecessary and elaborate laboratory examination.
Fewer examinations where negative findings are quite certain.
Elimination of fads and expensive overhead for show. Hospital
profit should go to paid clinical men in the place of increased
help, as required by the American College of Surgeons, to keep
up an unnecessary paper work. This is a hangover from the
government service and an emergency system of records is in
order that requires less clerical help.

In recent years the governing forces have placed severe dis-
cipline on medium education in the rapid raising of standards,
which was most necessary until now it has swung the pendulum
too far and crowded the profession with the scholar, the memory
fiend and the scientist, to the detriment of the plodder, the less
emotional and more stable individual who excels in the art of his
profession. With the removal of the apprentice and preceptor
something has been lost and not replaced.

The abuse of low standards and the commercial era wherein
460 medical schools came into existence produced an intolerable
condition, and regulating bodies, such as the American Associa-
tion of Medical Schools, Council of Medical Education and the
Federation of State Boards, have proved of untold value in estab-
lishing better trained men. Meanwhile, however, the country has
been flooded with great numbers of osteopathic chiropractors and

cult doctors, who were given legislative authority to practice, and
an abuse that was bad, and yet within our control, passed out to
the other bodies more sincere for financial gain, and less inter-
ested by basic or fundamental scientific training to carry on the
Hypocratic Oath.

The great problem of medical economics of the present hour
is a satisfactory distribution of medical service at a reasonable
cost, based on present monetary value. Just as the rise in wages
is lagging while the cost of commodities is soaring, so our scien-
tific advancement of medical knowledge, expensive in the appli-
cation, is far in advance of the actual need of our people. Thus
many communities have far outbuilt the needs of the people with
hospitals and service that would be required by a doubled popu-
lation ten or twenty years ahead of the times. It is impossbile to
meet the taxes and overhead and difficult to curtail with sufficient
rapidity to prevent great financial loss.

We must fight to maintain the present American plan with
proper readjustment and not turn over to state medicine and
paternalism the principles of independence and the care of the
individual with the family doctor until a financial balance has
occurred.

We cannot legislate these matters overnight, but by education
and concerted action a balance will be restored. Mass production,
industrial medicine and surgery are with us, and efforts to stand-
ardize methods for people rather than individuals are at work.
The applications of mechanical and electrical methods and use of
numerous laboratory aids, some expensive and of little value, and
a failure to evaluate and practically apply the personal equation
of the patient, the physician and specialist, have proven expensive,
misleading and often with unsatisfactory results.

The great public are now in a negative phase. They shy at
suggestions for diagnosis and treatment that are unnecessary, ex-
pensive and without certain financial return. They have taken
X-rays, had their blood examined, their metabolism and blood
pressure taken and many are no better. They are not satisfied
with the German method, much diagnosis and little or no treat-
ment. This school of medical scholars who maintain a beautiful
theoretical and fascinating study that costs the taxpayer in hos-

pitals and clinics, millions of unnecessary money must give way
to the public demand, temporarily at least, or the public will turn
more and more to the rubbers, the prayers, the patent medicine
man, and the soothsayer for satisfaction and advice.

You cannot suddenly change a people with tradition and habit,
with craving, when ill, for something to take and something
to apply, and substitute words, fine beds and buildings, scientific
lectures and promise, without creating, as we have, a restless,
suspicious and uncontented people, who wander from doctor to
doctor, from hospital to hospital, from specialist to specialist, to
satisfy a longing for relief from pain, fear or a desire for mental
and bodily satisfaction that all is well and that we are doing the
best we can.

If the leaders will not recognize these fundamental causes of
our inability to hold the American public, all or much more, is
lost. The re-establishment of balance must be based on restored
confidence.

The subdivision of labor has gone beyond its legitimate require-
ments, as these observations will show. The psychology of the
friends and relatives coming into the picture and recommending
their doctor has always been and always will be difficult, but today
practically every case is interfered with before the plan has been
under way three days. Patients are moved from hospital to hos-
pital, from doctor to doctor, with alarming rapidity, and cases
that are not operated upon the first day are difficult to handle and
to hold, even those who do not pay. Untold harm and loss of
confidence have come from the system of allowing any surgeon
to do any major operation without special qualification.

A partial remedy for this difficulty will be worked out by the
confederation of special examining boards and advisory councils.
Curtailment by the public of expense for quacks and nostrums
should be given greater emphasis by all national societies. The
abuse of unnecessary laboratory and X-ray examinations, over-
expensive rooms and special nurses and anesthetists. The profes-
sion should demand adequate care at a just price for indigent
sick, as most city charters provide, without luxury or waste.

Group practice, a response to public demand for special service
at reduced cost, has come into the picture. Institutions of this

kind have overgrown until personal supervision and responsibility have been impossible, and the personal equation, knowledge of the individual, interest in his welfare, are replaced by mass production methods of the assembling plant of an auto factory. Groups should be penalized for contract practice.

During the last ten years the graduates have exceeded the deaths by 832 a year; at present, 1,800 to 2,000 a year. American students studying abroad and returning for practice increase the crowding several hundred a year.

Some compensation by tax appropriation subsidy might make it more attractive for well-trained men to seek a rural location. Extra-mural courses and postgraduate work for specialists should be borne by teaching institutions with a minimum fee and decreased cost all down the line.

It is estimated that we have more than 6,000 who lay claim to otolaryngology as their choice of special work. If the specialties under a federation and advisory council would present a united front and join organized medicine in general, many readjustment problems would be readily solved. Support and loyalty, coupled with understanding and interest, can accomplish much toward economic good.

Society from time immemorial has formed into two grand classes: the constructive and the destructive. The building up of fundamental principles takes years and many workers to accomplish well-laid plans. We have been living in a destructive period, with panic, pandemonium, anxiety, hysteria, restlessness and loss of confidence. The "new deal" is under way for a restoration of things stable and standardized. An orderly retreat is necessary to establish reforms in our profession, our hospitals, medical schools, and fee systems, that will meet the general requirements of our people.

The destructionists, revolutionists and pessimists must stop, look and listen, like my old horse at the railroad crossing. They must recognize that the people should be held in confidence with our system of specialization and medicine in general by readjustments that are demanded by the times.

I uncovered an old Wayne County fee table of the horse-and-buggy age, in vogue some thirty-five years ago, and I believe that

we are in that period of deflation at present which will require that county and special societies give this matter careful consideration in committee assembled and revise a schedule that applies with justice to all throughout our various sections of the country.

This subject has been tabooed in the past with reason, for we lived in a different world and era. Business methods may be dignified, just and without hard-boiled features. They require more thorough discussion and general understanding, and discuss we must.

My early training in general practice, when calls were two or three dollars a visit, called for the receipt of one dollar or more to meet financial conditions, but invariably the bill was sent for two or three dollars with the discount given for cash or prompt payment. It was easy in that way to establish for your profession and yourself a just fee as the value of your service increased.

At this time the people recognized the value of skillful service, age, experience, plenty of time for them, interest, kindness, sympathy and attention. Shopping for an operation was rare, and confidence in the individual practitioner or specialist or doctor operated in the home, and we knew how and where the patient lived, and we could judge our fee accordingly. A return to home operating is untenable, but be must substitute a better knowledge of the patient .

Now, with the hospital frequently as the only surgeon remembered by the patient, with state medicine caring for thousands of people, with a World War and 6,000,000 soldiers given skilled care for nothing, is it surprising that our present condition exists?

Remedies must come thick and fast. We are good at therapeutics, but poor business men, wild in our investments, and given to criticism for those who show apparent gain. Yet remedies for our economic disasters must be formulated and a new economic deal must apply to our profession.

I believe that our profession should form an economic congress to discuss our own necessary reforms. This could easily be brought about at some central locality like Detroit or Chicago, or at the American Medical Association. It should perhaps be financed by some foundation fund or the American Medical Asso-

ciation, as was the committee on medical costs. Its delegates should include a complete cross-section of the entire profession.

The problems for study and discussion that require definite and quick solution would be recommendations to the family doctor and specialists in regard to fees, hospital charges and overcharges. Abuses in clinics and pay for clinical assistants. Subsidizing all private hospitals and physicians on attending service, including the staff. Decrease in beds built by the government, state, municipal and county, and relief to all hospitals classified by the College of Surgeons. Financial relief to standard laboratories that could be used by hospitals for government, county and city patients.

The use of proper private hospitals for government cases is cheaper, more efficient and of better educational value. It stimulates the pride of the individual and family on the need of independence and paying your way. A tuberculosis hospital, under my direction, had one-third full or part-pay cases until the State took over the burden in its entirety.

Government cases should be accepted on a part-pay basis if the private hospital could be guaranteed a partial payment plan. The taxpayer cannot keep up this burden. He is ruined at the present time with terrific welfare expenditure. His medical outlay must have quick relief. Many government hospitals operate at a cost of seven dollars or more a day while private institutions can do the work at four dollars under the present value of the dollar.

Under this system of government relief, by closing our great state hospitals, by encouraging those who could pay for service, on a part-pay basis, subsidized by the state, more of our people would return to the family doctor and specialist. Each hospital subsidized would pay the attending staff as other employees and the wage of all hospital workers could be increased.

The medical school curricula may be changed to allow a maximum of five years for college and medical training in A class schools for those who would enter general practice. Those who chose special fields would enter schools for specialists requiring two or three years more.

A confederated Advisory Board of all specialties will soon pass on the requirements and standardize the same. The effect on medical education will then be far-reaching and revolutionary

throughout the country. The great medical centers will then be classified, with two or three or more years in our universities, where the eye, ear, nose and throat will be taught in the same thorough way as at present but much more extensively. The specialist's course would include all allied subjects, such as borderline pathology, surgery, medicine, diseases of the chest, neurology and roentgenology.

It is difficult to believe that the words of the medical cost committee that run in places like the babbling brook will stampede a stable section of otolaryngologists to grave anxiety or alarm. Common sense, education, judgment and a study of the problem tell us that medical costs will adjust themselves as they have in previous decades under supply and demand, the cost of commodities in general and wages. We do not need the tingling brass economist out of the textbook to tell us what we know, and that is that no man, woman or child can spend two dollars for very long when his income is only one dollar.

We do not want our country to be like Russia or any other country but the dear old U. S. A., as she was. It was the best in the world, medically, specially, and every other way. The medical economics that will work out a return to the days of 1920 to 1930, or the good old days before the war, will be highly satisfactory, and they are coming just as sure as night and day, if we will not talk and lament too much, but work on the practical remedies that are necessary.

It has been said that the labor of five years by the Committee on Costs of Medical Care, as a mountain, has labored in vain— it has brought forth a mouse. The report, to be successful, must give us a practical, workable plan that will allow our profession to live and the laity to profit. State medicine is not the answer nor has insurance in other countries, or ours, elevated, dignified or improved the financial status of the specialist. Taxation pays 14 per cent of the bill for medical care, and an overburdened people with millions of delinquent taxes cannot continue without disaster.

A bureaucracy in medicine is not desirable, as our present economic system has found it necessary to curtail demands for tax money in the Army, Navy and public school system.

If the public health departments would return to the police function as originally intended and care for infectious and contagious disease and turn aside to the specialist—eye, ear, nose and throat; to the general practitioner, obstetrics, pediatrics, cancer and syphilis, the flow of cases for pay or part pay would reestablish the family doctor on a much more profitable basis. This can only be accomplished by the economic stress of the present used to adjust appropriations and lower taxes.

The necessary hospitals and clinics are those for teaching and emergency. It is only necessary to visit the hospitals of the old country, in the British Isles and on the continent, to realize that many of those buildings, hoary with age, have turned out some of the most brilliant and scientific results of the country.

The family doctor must come back to reduce medical costs and the specialist must work with him as ever for such compensation as the case affords.

We want no increase in state medicine, in bigger or more expensive municipal hospitals; we want no compulsory insurance system or cheap health insurance associations, such as they had in Cuba and many foreign countries. Mass production of health by schemes of big business men must be laid aside, but the people must pay the doctor for his illness as in days of yore. Pay in commodities or work, if you like, but pay he must, the doctor and the specialist and the hospital, a just and readjusted fee commensurate with the service, modest with a small measure of profit that will allow a wage equal to and better than the policeman, the fireman, or the small merchant, whose investment in education or good is usually small indeed.

Our specialist must live with a budget of retrenchment to meet these new conditions. His automobile, his house, his office, must be smaller. His wife will share his new burden with the same joy and thrift as of thirty years ago. As the neighbors are doing it, the task is easier.

Many of us fail in these hard times from a lack of proper business methods. Our societies seldom deal with this essential of success, and our medical schools fear to discuss the problem for fear that commercialism with the misinterpretations follow them.

The practical solution of life must ever be that to do our work for others, our legitimate compensation must be sufficient. Proper business management should be a part of our curricula and with it courses in bookkeeping, money and banking, elementary law, and more psychology and salesmanship.

If you believe in your operation as a genuine and necessary aid toward the recovery of your patient and you have confidence in your ability to perform the same, is it not a dignified and worthy part of your education to sell that operation to the patient? The man who does this part of the job well is usually the chap, I find, who does the operation whole-heartedly.

How can you learn these things in the welfare clinic, the municipal hospital or the Board of Health? These honest, legitimate methods of promoting a genuine benefit to your patient must be paid for if economic success is to be maintained.

Why is one man a success and another a failure? The causes may be many, but the man who has personality, common sense, knowledge and love of his profession, and an understanding of his people, a recognized honesty of purpose, a true interest in humanity, a sympathy for suffering, energy and a love of medical study and ability to fall into a difficult situation, who knows and who is not afraid of work, will win every time.

We have heard much of the forgotten soldier, the forgotten man and woman, but is it not true that in this economic earthquake there is a forgotten doctor, and the doctor forgets the specialist?

We were generally allowed to appear last when debts were paid, but today we are forgotten entirely by many people. The banks, railroads and farmers are given life sustaining appropriations. We ask for none, but in the appeal to fairness is it not just that state medicine halt in its onslaught with the taxes of our people, and that in tax reduction a share be used in subsidy for the specialist and the doctor to meet many but not forgotten bills?

The new economic state of mind of thousands of people under bad education of the World War, extensive charity, with no questions asked, the efforts of Boards of Health to sell their goods and hospitals free, has ruined the desire to be independent among a vast number of people who formerly considered it a disgrace

to ask for charity for themselves or their family while any available money remained in the family exchequer. The new economic era has completely modified this idea. Many consider it a right as a taxpayer to demand government or free care. The abuse has spread to the ex-soldier, who demands liberal compensation for all non-connected disabilities. Vast great hospitals have cared for thousands of men who could well pay for service. The otolaryngologist has received government appointments without necessary qualifications. An enormous amount of free work on the ear and throat has been done.

The remedies for these abuses are at work, and the College of Surgeons should require at least government hospital appointments in otology from those who have been certificated by the Board.

The restoration of human values is the problem before the medical profession. The rebuilding of character and the establishment of honor instead of dishonesty, which is rampant everywhere. The word and promise of a man must be good again before we can hope to readjust on a sound basis. Religionists and doctors and otolaryngologists must unite in a grand crusade and organize with propaganda that will return to the world the lost individual character. Carloads of words on economics and politics have destroyed religion and undermined the true teachings of Christ, which was and is character, honor, the duty of the gentleman, a defense of the Preamble of the Constitution of God and country, and do unto others as you would wish them to do to you. These fundamentals of ethics, of economics and of human relations must be restored for the otolaryngologist and the world.

The remedies I would advocate are:

1. A complete readjustment of our fee system to meet the changing value of the dollar.

2. Greater devotion to organized medicine and the specialties to improve our feeble power in the control of state medicine.

3. A rapid readjustment of the budget of expense in hospital, office and living expense.

4. Stop the building of new hospitals, additions and medical schools until the overflow and waste can be checked.

5. Revise medical costs in nursing and hospitals to conform with supply and demand.

6. Teach the public the value of service by the certified otolaryngologist as compared with the untrained man.

7. Recognize and encourage postgraduate study in qualified centers in our own country.

8. Return to U.S.P. and N.F. for the use of drugs and solutions.

9. Promote the plan of a paid attending and assistant staff in medical schools, hospitals and clinics.

10. Advocate the teaching of bookkeeping, elementary law, money and banking, major psychology and the study of human relations in the premedical course.

11. Regulate state medicine to a police function and the care of infectious and contagious disease, and return all pay cases to the family physician, the qualified specialist and the private hospitals, all of which are now running at one-third capacity while the taxpayer attempts to pay the bill.

12. Cut the appropriations of university and municipal hospitals to the number of beds required for teaching or emergency only.

13. Pay all teachers in medical schools an honorarium or better.

14. Pay all school and outpatient department physicians and specialists the cost of service or better.

15. Eliminate contract and industrial medicine except for emergency service, and allow the choice of physician or specialist to the individual.

16. Elimininate U. S. Government hospitals and the staff except for service-connected disabilities.

17. Allow only qualified specialists to serve as such in Army, Navy and Veterans' Bureau hospital centers.

18. If the earning power of the otolaryngologist is to increase, it must be along the lines of borderline surgery and better general medicine.

19. Have the county society vise all contract practice.

20. Let the county and special society give more consideration to economic subjects.

21. If we would restore the confidence of the people, fee splitting must be stopped by more drastic methods.

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XIX.

HYPOTHYROIDISM AND VASOMOTOR RHINITIS.

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While allergists have been busy establishing the allergic nature
of most cases of vasomotor rhinitis, other workers, the endo-
crinologists and the rhinologists, intrigued by the myxedematous
appearance of the tissue of the nose in these cases, turned to thy-
roid deficiency as a possible explanation for the mechanism under-
lying vasomotor rhinitis. In their minds, this was confirmed by
the fact that many of these patients did show a basal metabolic
rate that was below the average and that some of these were re-
lieved by the administration of thyroid preparations. It is the
purpose of this paper to correlate these facts with the concepts
of allergy and to offer in this way an explanation for the success
as well as the failures with this method of treatment.

"Allergy" is used in this paper in a strictly technical sense as
covering all forms of specific hypersensitiveness which have been
found to date in the human. "Atopy" is used to mean that form
of allergy which is controlled by inheritance in contradistinction
to the other types of allergy, such as serum sickness, contact der-
matitis, bacterial allergy of the tuberculin type.

Atopic coryza is that form of vasomotor rhinitis which is
produced by a specific, inheritance controlled hypersensitiveness
usually to proteins. This atopy is the primary cause of the coryza.
As in all forms of atopy, there may be operative at different times
secondary factors which aggravate the condition. The most com-
mon of these aggravating factors are barometric changes, tem-
perature changes, emotions, fatigue, malnutrition, infections, me-
chanical irritants and disturbances in the function of the ductless
glands.

These nonspecific aggravating factors may assume a rôle of
major importance in certain patients and in certain individual
attacks in all persons afflicted with atopic coryza. Of these, one

of the most important in certain cases is a deficiency in the secretion of the thyroid gland. Some physicians give thyroid gland preparations to all patients with a vasomotor rhinitis in the blind hope that they may be needed. Still more frequently their administration rests upon the finding of a lowered metabolic rate. It is not safe to assume that there is a deficiency in the secretion of the thyroid merely because the basal metabolic rate is below the average. The basal metabolic rate must always be correlated with the symptoms, the history of the case, the physical findings and the results of other tests. A lowered rate may result from a good many causes.

In the study of all persons presenting evidences of a vasomotor instability, it is important to evaluate the function of the thyroid gland, even though the patients are definitely atopic. For this purpose we should classify our cases of atopic coryza on the basis of our diagnostic studies as follows:

A. Those patients with an atopic coryza and normal basal metabolic rate. (This group is without the scope of this paper.)

B. Those patients with an atopic coryza and a lowered basal metabolic rate but who present no other evidence of abnormality.

It is assumed, of course, that the metabolic tests are carefully made and the machine has been adequately checked. In spite of the fact that most errors result in a reading which is higher than the true reading would be, still there are many instances in which the basal metabolic reading is low in cases which are not due to a thyroid deficiency.

Since individuals whose thyroid glands have been removed develop obesity, and since thyroid preparations can be taken to reduce the weight, the conception is generally established that thyroid deficiency is a principal cause of obesity. Such, however, is not the case. It is true that a considerable number of fat persons do show an apparently low basal metabolic rate. This is apparent but not real in the vast majority of instances, and comes about through an error in the computation of the test. Fatty tissue is metabolically inert tissue. Consequently, the basal metabolic rate should not be calculated, as is so often done, on the basis of

actual weight, but rather upon the ideal weight of the patient.
When this is done, the average rate in these fat people will be
found to be around 125 per cent of the normal. No one would
think of giving a patient with a basal metabolic rate of plus 25
per cent any of the thyroid preparations. Frequently these pa-
tients, on the basis of an improper calculation, are given thyroid,
both for the obesity and their vasomotor rhinitis. Weight reduc-
tion through carefully planned diets in which there is an over-
abundance of vitamins, and not thyroid, is indicated in these cases.

It has been shown that this rate is lowered appreciably after
prolonged subsistence on a strictly vegetarian diet. The inade-
quate war time diets of the Europeans were also found to lower
the rate more proportionately than it did the body weight. This
was confirmed by the studies of Benedict, whose young men on a
semistarvation diet lost less than 10 per cent of their weight but
at the same time showed a 16 to 20 per cent lowering of their
basal metabolic rate. There is now considerable evidence to show
that the habitual consumption of food by the present day college
woman is lower than it was a generation ago. While the calories
of the average diet of these women was not adequate, many of
these diets which have been analyzed are still more deficient in
their proteins than their caloric content. It is therefore apparent
that undernutrition is an important factor in many instances of
a low metabolic rate. This is especially true of the young woman
of today who, because she desires to remain slender, subsists on
a ration which is barely within the lower limits of adequacy.
Sooner or later these undernourished girls, because of their activ-
ity, become nervous and easily exhausted. Because of these symp-
toms, the basal metabolic rate is determined and frequently thy-
roid is prescribed on the basis of a finding below the average. It
is, of course, not the best practice to stimulate a starving and
therefore exhausted body to further activity. In such instances,
the use of thyroid over any considerable time may do much harm.
Where the appetite has been lost, some physicians give thyroid
for a short time only in an effort to bring it back. Certainly these
patients do not need it as a treatment for their atopic coryza.
If such be present, these patients are undernourished and their
bodies are calling for food. The lowered metabolic rate is simply a

conservation effort to protect them. The adequate feeding of a properly balanced diet is the best, nonspecific measure in the treatment of their nasal condition.

In children from 8 to 14 years of age, the basal metabolic reading is frequently as low as minus 20 per cent, without any apparent evidence of a disturbance of any of the glands of internal secretion or any disease at all for that matter. The explanation for this observation lies in the fact that the figures used for the normals in the computation are too high because they were determined on boy and girl scouts in exceptionally good physical condition. We should therefore consider readings as low as minus twenty in children as being low normals, rather than abnormally low determinations.

C. Those patients with an atopic coryza and a lowered basal metabolic rate, who present some evidence of abnormality.

It is most important to recognize among the patients with atopic coryza, a group of them who are constitutionally asthenic with the symptoms complex of autonomic nervous dysfunction, although the nature of the dysfunction is by no means the same in all cases. The constant factors of low blood pressure, a low threshold of emotional stability, failure to gain in weight, or a loss in weight, a sensitiveness to cold, and often a diminished catamenia, lead to the finding of a low basal metabolic rate. These patients, however, do not do well on thyroid medication. Clinically, in this group, it is possible to rule out malnutrition, starvation and hypopituitarism. All the patients are probably suffering from some sort of endocrine disturbance and should be studied from this viewpoint. Blumgarten has offered chronic adrenal insufficiency as a possible explanation and there is considerable evidence to support this theory.

Second, under this classification are those cases in which the outstanding abnormality appears to be an irregularity of menstruation (always scantiness or temporary amenorrhea, or both).

Third are those who satisfy the strict requirement for a diagnosis of hypothyroidism. We have three methods for determining a deficiency in the secretions of the thyroid gland. In the order of their application they are:

1. The detection of the clinical symptoms of hypothyroidism, which are—

(a) A typical dry, pale, edematous skin which does not pit upon pressure and which often has a muddy, jaundice-like tint with brown splotches at times. These trophic changes are apparently variable but the dryness of the skin is the most constant. The scaly roughness of skin of a true myxedema is not to be expected in these cases of milder hypothyroidism. If the skin about the elbows, the knees and on the extensor surfaces of the body, however, be examined, it will be found to be involved in a high percentage of instances.

(b) The hair often presents a dryness and sometimes in the more advanced cases a coarseness, but this is by no means constant. In the milder cases, one may notice a slight thinning of the outer margins of the eyebrows and a loss of luster to the hair of the scalp. Not infrequently women will observe that their hair has become brittle and breaks or splits easily, while men may observe that the beard is not so heavy as it was formerly.

(c) The finger and toe nails sometimes showing an increased brittleness.

(d) Dry and swollen mucous membranes, and this is difficult to distinguish from the appearance of these structures which are produced by allergy.

(e) An anemia, usually of the secondary type, may be present.

(f) A consequent retention of the barium meal in the descending colon and sigmoid for 56 to 72 hours, in a high percentage of instances.

(g) Quite constant increased mental sluggishness. Atopic persons are, as a rule, above the average in intelligence and alertness. It is only when the picture is complicated by a hypothyroidism that these patients begin to have difficulties with office and school work. Thoughts come slowly. The memory is poor and there is a lack of interest. Occasionally melancholia and hallucination psychosis develop.

(h) Rheumatic pains in the joints, especially in the spine.

(i) In the female, menstrual disturbances and sterility.

Hypothyroidism is an important causal factor at times in amenorrhea and more frequently menorrhagia, and probably abortion, miscarriage and premature labor, and death of the fetus. Hypothyroidism is one of the most frequent causes of menorrhagia and metrorrhagia and should always be excluded before resort is had to the curette, radium, X-ray or the knife.

(j) In the male, a loss of libido and impotency. Spermatorrhea may be an early expression of hypothyroidism.

(k) All variations of sensitiveness to cold. In the extreme cases, it is highly characteristic and consists of an unbearable chilliness of the entire body. The patient ordinarily wears extra clothing, uses more bed covering at night than the average person and strenuously objects to a room temperature which is comfortable to the healthy members of the family. In the milder cases, the sensitiveness occurs only as "constantly cold hands and feet."

(l) Lowered blood pressure and slow pulse.

(m) Lowered resistance to infections of all kinds. Frequent colds, coryza and respiratory infections are common and several authors have emphasized the frequency of gall bladder trouble. Here the history must not be accepted at its face value, but the examiner must assure himself that these symptoms are not a part of the atopy underlying the coryza rather than true infections of the nasal mucous membranes.

(n) Fatigability, "lack of pep" and endurance, and a feeling of being tired all of the time. These patients find it difficult to keep up with their daily tasks. A night's sleep does not refresh them. They awaken tired in the morning. They may work up toward a peak of energy some time during the day, but it does not hold and they are exhausted by night. In this, they differ from the neurasthenics, who brighten up as the evening approaches.

2. The application of extensive and systematic laboratory procedures. Here the methods of Rowe are of great value in those cases in which it can be employed.

Basal metabolic rates running less than 90 per cent of the expected rate may be due, in addition to the hypothyroidism, to a de-

ficiency of the pituitary gland or to its dysfunction, to a deficiency of the ovarian secretion or to a diminished function on the part of the adrenal glands. While it is true, as a rule, that rates of less than a minus 25 per cent are usually due to a thyroid deficiency, such is not always the case. The D-galactose tolerance test will prove of considerable aid in making the differentiation.

3. The therapeutic test. When it has been determined with a reasonable certainty that the patient does have hypothyroidism, he should be given thyroid preparations. These are usually referred to as extracts, which they are not. Thyroid U. S. P. calls for not less than 0.17 and not more than 0.23 per centum of iodin. Different manufacturers measure the strength of their preparations differently. In order that there be no confusion, the strength of the preparation should be stated. As Harrell has said of the instructions to give from five to fifteen grains of thyroid daily, "it would have been just as accurate for him to have prescribed ten grains of purgative daily—the druggist would have known just as well what he wanted." It is a good rule to get a preparation and stick to it so that you will know what it will do. A patient with hypothyroidism will respond definitely and promptly to the administration of thyroid, and the other cases presenting somewhat the same picture will not be influenced favorably. Instead, such medication may produce in these patients the symptoms of an overdose, such as a fast pulse, weakness, tremors. Occasionally all of the thyrotoxic symptoms except the rapid pulse will be induced by the use of thyroid in a case with a low basal metabolic rate which is not a case of hypothyroidism.

4. Those cases with atopic coryza and a basal metabolic rate below the average, with various conditions not included in the preceding three groups.

These are identified by elimination and the methods outlined in this paper. They will be found to be due to starvation, pituitary disorder, ovarian or adrenal deficiency and muscular atrophy.

The foregoing may therefore be summarized as follows: If all cases of vasomotor rhinitis are studied from the viewpoint of allergy, we shall find that most of them are atopic in nature. The recognition that this is true in the individual case rests upon four criteria, viz., a family history of atopic manifestations, a

personal history of the same, an eosinophilia both in the circulating blood and in the nasal smears, and finally positive skin tests for protein sensitization. Of the cases identified as atopic coryza, a certain percentage are aggravated by the existence of a deficiency of thyroid secretion in the patient. These can be suspected by the clinical symptoms of hypothyroidism and can be identified by a basal metabolic rate determination plus a galactose tolerance test and the routine laboratory studies. All this can then be confirmed by the therapeutic test. In this way no harm is done but good can be accomplished, for the use of the thyroid extract is an adjunct of great service in cases which have been selected in this way.

394 EAST TOWN ST.

Clinical Notes and New Instruments.

XX.

BRANCHIAL CYST: TWO INSTRUCTIVE CASES.*

GORDON BERRY, M. D.,

WORCESTER, MASS.

INTRODUCTION.

These two cases are reported because of their scientific interest as embryonic anomalies. The first showed two symmetrically placed cervical fistulous tracts, remnants of the second visceral clefts. The right side had healed when this case came under my observation; the left side drained pus into the left tonsil fossa until the radical dissection and removal of a large cystic sac cured the trouble. The second case, though unilateral, closely parallels the anatomy of the first and is added for its supporting evidence. Interestingly there was an "embryonic rest" at the lower end of this fistula.

EMBRYOLOGY.

In the human embryo the visceral arches are best developed in the last half of the third week. In fishes there are six of these arches. In man, these are reduced to five parallel cartilaginous bars with intervening furrows, the fifth bar, however, blending with the surrounding structures. The gill clefts of the fish are filled in in man by occluding ectoblastic and entoblastic membranes, giving us external grooves (visceral furrows) and internal grooves (pharyngeal pouches).

Arch I (the mandibular arch) differentiates into an upper short process forming the nasomaxillary bones, and a lower, longer process forming the lower jaw and floor of the primal oral cavity.

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*Read before the American Laryngological, Rhinological and Otological Society, in Chicago, June 10, 1933.

Arch II (the hyoid arch) forms the lesser horn of the hyoid, the styloid process and ligament and the stapes.

Arch III (the first branchial arch) forms the body and the greater horn of the hyoid.

Arches IV and V (the second and third branchial arches) fuse into the thyroid cartilage.

Of the external grooves (visceral furrows) the first remains as the external auditory meatus. The others disappear by the twenty-eighth day, excepting an occasional remnant in the lower lateral part of the neck called the sinus praecervicalis of His. If the occluding membrane breaks through, this sinus becomes connected with the pharynx as a cervical fistula.

The internal grooves or pharyngeal pouches remain: I becoming the eustachian tube and middle ear cavity; II disappearing except where it contributes to the epithelium of the faucial tonsil and the supratonsillar fossa; III and IV form the thyroid and the thymus.

CASE HISTORIES.

Case I—G. D., a strong, healthy, heavily-built worker in a shoe factory, born in Italy 34 years ago, recalls as a child a small hole on each side of his neck from which thin secretions would periodically escape. At twenty years of age a "country doctor" cauterized and treated with iodine these discharging sinuses and they healed.

Present Illness.—For the past two months the patient has noted a swelling in his left neck. Pressure on this reduces the swelling and pus escapes into his throat. There is no fever and no pain, only some discomfort from the swelling, relieved when the pus is expressed.

Fig. 1 shows the soft swelling on the side of the neck. Case 1.

Fig. 2 shows the bilateral lifting tug of the branchial cord remnants during the act of swallowing. Compare with Fig. 1.

Fig. 3. Roentgenogram of branchial cyst; sac injected with lipiodol. The shadow extends up to the tonsil fossa.

Examination on September 27, 1930.—Externally a faint, small scar appears on each side of the neck at the level of the first tracheal ring which becomes well defined as two symmetrically placed dimples during the act of swallowing, these scars being apparently pulled up by cord-like fibrous processes. Internal to the left sterno-cleido-mastoid muscle is a long cylindrical swelling, deeply situated and passing up from the cricoid to above the hyoid level. It is soft on palpation. The part of the examination which further concerns us is the tonsils. They are large, congested, showing chronic cryptic disease. In the left plica supratonsillaris is a red, soft swelling or papilla, and pressure on the left neck swelling causes thick, creamy pus to escape through a small opening in this papilla. A small probe can be made to enter but a short distance (4 to 5 m.m.).

Diagnosis and Course.—The diagnosis made was a right branchial sinus, healed; a left branchial cyst and internal fistula; a chronic tonsillitis. The removal of these diseased tonsils and then of the cyst was recommended.

At the time of the removal of the tonsils a month later (October 22, 1930), I was able to establish that the fistulous tract passed through the left plica supratonsillaris and over the upper pole of the tonsil, apparently
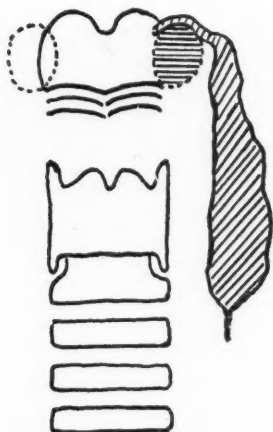
Fig. 4. Schematic drawing of branchial cyst and its relation to the tonsil. It is seen piercing the superior constrictor muscle, passing up over the tonsil in the peritonsillar fibrous tissue, to empty into the mouth through the plica supratonsillaris. The lower end was attached to the skin opposite the first tracheal ring, by a fibrous cord. Sac, ½x4 inches.

running in the fibrous peritonsillar tissues. About one-quarter of the way down the fossa from the upper pole, the tract turned outwards and pierced through a small cleft in the superior constrictor muscle, then passed down into the neck tissue.

The tonsillectomy convalescence was uneventful. Pus continued to escape from the fistula in the left tonsil fossa, but drainage was freer and he felt better, so he did not return for two years.

On June 7, 1932, preparatory to removing the cyst, I injected lipiodol into this fistula. The roentgenogram showed the cystic sac to extend from the second cervical down to the sixth cervical vertebra, smaller above and one-half an inch across in the lower part.

Operation.—Under avertin by rectum and novocain locally, the cystic sac was readily removed intact through a semilunar 5-inch neck incision. It was found just in front of the carotid arteries, its lower end superficial and attached by a short fibrous cord to the neck scar at the level of the first tracheal ring, the upper end passing deeply till it could be felt by palpation to go through a small ring-like opening in the superior constrictor muscle of the pharynx. Blunt dissection of this pedicle as it passed through to the mucous membrane surface of the tonsil fossa (preparatory to applying a purse-string ligature) made it tear away at this inner upper point. The cyst was four inches long and one-half an inch in diameter.

Fig. 5. Front view of branchial fistula, injected from
below with lipiodol. Case 2.

A small drain was left in the closed wound for six days. Convalescence
was uneventful; the tissues closed off completely without further trouble.
There remains in the upper part of the left tonsil fossa a small superficial
pocket 3 m.m. deep which looks clean and healthy, evidently the extreme
upper end of the original fistula.

Case II—F. F., an 11-year-old boy, was admitted to the hospital three
weeks ago (May 19, 1933) for a soft, fluctuant, gland-like swelling low
in the left side of the neck, of five days' duration, which proved to be the
lower end of a branchial fistula. The other end emptied into the upper
part of a scarred left tonsil fossa from which the tonsil had been
removed.

Course.—At birth the mother noticed an opening low down on the pa-
tient's neck. When nursing, milk would flow through this opening. At
three years of age, a little pus began to drain here. At five years, the
tonsils were partly removed and this sinus was "probed and treated."
Apparently this sinus then healed and remained closed. In 1930 what
looked like a broken down tubercular gland was removed from just above
the midclavicle. When sectioned, this proved to be an "embryonic rest"

Fig. 6. Side view. The opaque fluid in the fistulous
tract can be traced upward and forward to the tonsil fossa
area, where it emptied into the pharynx.

containing blood vessels, nerve tissue, striated muscle and connective
tissue. The wound was sutured and healing resulted. Just prior to my
first seeing him, this old area low in the left neck had again swollen, five
days previously. Aspiration of the infected fluid and the injection of
lipiodol (by Dr. Armand Caron) permitted the roentgenogram demonstra-
tion of a cystic area at the bottom and a thin tract ascending to the left
tonsil area. Then the cystic area was incised and drained and methylene
blue injected. This escaped into the pharynx from a small vent high in
the scarred tonsil fossa.

TREATMENT.

A modified Carnoy's solution was injected from below as an
escharotic in the hope of avoiding the extensive scarring of an oper-
ation. Another injection may be necessary. This procedure is recom-
mended by Elliott C. Cutler and Robert Zollinger of Boston in the March
number of the American Journal of Surgery. (See also the experimental
work on cysts in the brains of dogs by R. Zollinger and A. R. Moritz in

the Archives of Neurology and Psychiatry, November, 1932.) The solution is made up of absolute alcohol, 6 c.c., chloroform, 3 c.c., glacial acetic acid, 1 c.c., and ferric chlorid, 1 gram. It apparently acts somewhat similarly to Zenker's Fluid, but causes less pain. Care must be used to protect the skin and mucous membrane against burning.

CONCLUSION.

We are dealing here with remnants of the second visceral grooves or clefts. The first case is of peculiar interest because of the bilateral symmetry of the original neck opening, which had closed. The second case had a fairly large "embryonic rest" (2.5 x 1 x 5 cm.) at the lower end. Both cases demonstrated accurately the course of the tract as it passed upward into the deeper tissues to finally discharge into the upper part of the tonsil fossa, if the tonsil be removed, and up over the tonsil and through the plica supratonsillar, if the tonsil be present. The modified Carnoy's solution injected offers a new and easy means of obliteration which seems logical and may prove useful in other phases of our work as well as here.

36 PLEASANT STREET.

XXI.

LINGUAL THYROID.

F. J. BISHOP, M. D.,

SCRANTON, PA.

In a search of the relevant literature the incidence of lingual thyroid has been revealed to be uncommon, overlooked, not recognized or diagnosed.

The fact is that many of the large clinics report only a few cases, and, as Ziegelman[a] suggests, "Some surgeons may not see more than one or two during their careers." New[b] of the Mayo Clinic reports only ten cases.

It is fair to assume that all of these cases are not reported: (1) in recent literature, after attention had been called to the condition, more cases have been reported than in the preceding greater number of years; (2) the condition can be and is present in the newborn, frequent in childhood and more frequent in young adult and middle age, less common in older people; (3) the same results were obtained under similar conditions; as for example, abscess of the lungs, following operation on the upper respiratory tract; (4) the presence here of usually a part of the gland results as a failure on the part of nature to complete the growth downward of the diverticulum to the normal location.

For these reasons I desire to add one case to the present list of 130, a very meager number in comparison to reports of other anomalies.

The embryology[1] will be omitted because this has been reported in the more recent articles, and is available in any of the standard textbooks.

The classification of types and location given by Lahey[2] are as follows: (1) "Those that remain and develop at the point of fetal origin, the foramen cecum, and are clinically known as lingual goiters; (2) those that develop and remain localized in the

structure of the tongue called intralingual goiters; (3) those developing in front of the larynx, the prelaryngeal type; (4) those that develop and assume the normal position anterior and lateral to the upper rings of the trachea, known as pretracheal; and (5) those that develop in the superior mediastinum behind the sternum, commonly called retrosternal."

History of Case.—Mrs. E. C., aged 31, white, housewife, mother of four children, was referred by her family physician for the following complaint: "Last night after eating some blackberries, I felt that a seed had stuck in my throat and was continually pricking." Family history irrelevant. Usual diseases of childhood; no severe illness; no operation.

Present Illness.—No complaints until the preceding night, as given in the above history. Never had noticed any trouble in drinking or swallowing, no pain; in fact, had never complained of any foreign body or new growth in the oropharynx. Later her mother, being questioned, said that she had noticed some change in her daughter's voice at least a year previous; the patient had not noticed this. In other words, nothing in her previous history would give any inkling as to her complaint.

Clinical Findings.—Upon opening her mouth a casual glance reveals a large mass in the oropharynx. Protrusion of the tongue increases its size; on depression of the tongue it is readily seen that this mass is connected to the base of the tongue by a rather large pedicle, to the left of the foramen cecum. It is impossible to see any deeper in the pharynx because of the size of the mass, and if not for the knowledge of the existence of the pyriform sinuses one would wonder how this patient was able to drink or swallow any size bolus of food. (Fig. 1.)

Physical Characteristics.—The mass consists of four or five lobulations, covered by a smooth mucous membrane, appearing similar to the normal membrane of the surface of the tongue. Five large blood vessels run from the base to the apex, anastomosing with similar vessels on the posterior surface. It is freely movable on manipulation, ascending and descending on deglutition.

Tentative Diagnosis.—Lingual thyroid.

Physical Examination at Hospital.—Heart and lungs are negative. A noticeable enlargement of the usual thyroid gland. Laboratory test: Wassermann test negative. Urinalysis: Slight amount of albumen; very few casts. Blood count practically normal. Hemoglobin about 85 per cent. Metabolic test: Plus 3 per cent.

Diagnosis.—The history, the metabolic test, the physical examination, the enlarged pretracheal thyroid and, in the oral cavity, the tumor and the presence of the uncommon pedicle (Harvey[f]) definitely locating the new growth slightly to the left of the foramen cecum made the diagnosis in this case comparatively easy.

The metabolic rate, the presence of all of the gland in the normal location, is imperative in our endeavor to decide the rather difficult question, Are we dealing with an aberrant or an accessory thyroid gland before the operation? Unless the symptoms are severe enough to demand its imme-
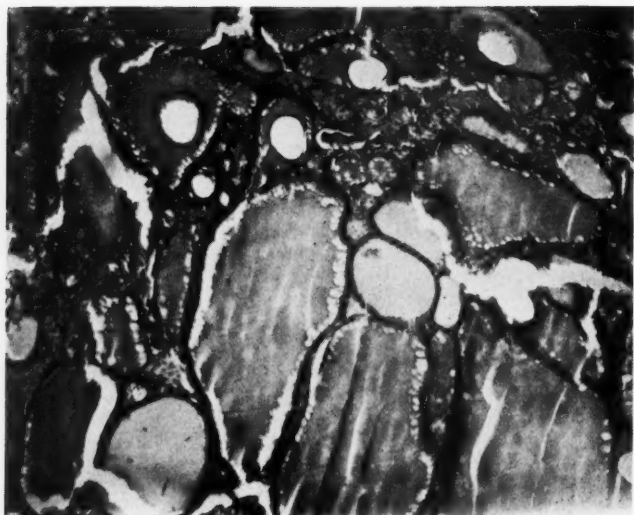
Microphotograph of lingual thyroid.

diate removal, every effort must be made to respect the above caution, otherwise the removal of the tumor will be followed by a train of distressing symptoms, namely:

Myxedema is the inevitable result if too much of the active gland is removed, if the removed part be of the pretracheal, aberrant or accessory gland; this will necessitate the continued feeding of the gland substance with fairly good results; occasionally the feeding fails to produce the expected results. Transplantation of the gland should be seriously considered.

Operation.—Patient seated in chair, head being held by nurse, patient retracted her tongue. Two applications, within a few minutes, of a ten per cent solution of cocain were applied to the base of tongue and around the pedicle, on a pledget of cotton held in a laryngeal applicator. After an interval of five minutes a Fielding-Lewis tonsil snare was used with the ordinary tonsil wire, except that both ends of the wire were threaded on the same side of the stylet, resulting in a wire loop at right angles to the handle. The loop was slid over the mass and was contracted, engaging the pedicle. Pressure was applied and maintained by thumb screw on the snare for about a minute when the vessels collapsed and the whole mass blanched. Then the pressure was increased and again maintained by the thumb screw. About two minutes later the pedicle was severed. The astonishing feature of the operation was the almost total absence of bleeding. Without further treatment the patient returned to bed and kept quiet until the next morning when she returned to my of-

fice. At this time an application of iodine compound was made and patient allowed to go home, returning to the office two days later. The result was a nice clean wound which, within a short time, could not be recognized from the surrounding normal tissue of the tongue. The patient was seen at short intervals, several times during the month with practically no complaints. Recovery uneventful.

The operative technic in this case is very simple compared to the usual operation and is applicable only in the presence of a pedicle. I cannot conceive how this simple technic could be used to remove the usual growth, which has a broad base. The operative procedure of the latter type of growth has been described in all the articles referred to in the references quoted, v. s., therefore refrain from repetition.

Pathologic Reports.—The results of the two independent pathologic reports are as follows:

Pathologic Department, State Hospital. Patient: Mrs. E. C.; lingual thyroid; October 30, 1932. Dr. F. J. Bishop, State Hospital, Scranton, Pa.

Tissue Thyroid.—The specimen weighs fifteen grams and is two and one-quarter inches long, one and one-quarter inches high and one and one-quarter inches wide. It is covered by a smooth shiny capsule. Cut sections present a smooth, shiny, dull gray surface, with encapsulated tumor in the center of it. Several small hemorrhagic areas can be seen on the cut surface.

Microscopic Examination.—Aberrant thyroid tissue showing inflammatory change. C. L. Mattas, M. D.

Patient: Mrs. E. C.; lingual thyroid; November 5, 1932. Received November 3, 1932; Dr. F. J. Bishop, State Hospital; No. 32-375.

Gross Pathology.—The tumor as received is bisected, each half being about one inch in diameter. There is a thin but distinct connective tissue capsule which has apparently been removed intact. The cut surface shows a dull gray surface with an encapsulated tumor about one-quarter of an inch in diameter near the center. There are a few small hemorrhagic areas and no cysts. The diagnosis from gross examination was not made. Thyroid tissue was not suspected.

Microscopic.—Sections of the entire tumor under a wide angle view magnified twenty-five times shows several irregular connective tissue septa running through the tissue and dividing it into more or less well defined lobules. One of these must have given the impression of a completely encapsulated mass noted on gross examination. Magnified one hundred and twenty times, practically all types of ordinary benign thyroid lesion are seen. In a few small areas the normal looking acini are considerably distended with diffusely pink staining colloid. In many acini the edges of the colloid are extensively vacuolated so that the gland seems definitely to have been functioning, at least to some extent. In some places there are closely packed masses of cells with little or no lumen showing the fetal type. A larger portion, probably the majority of the section, consists of rather dense connective tissue stalks covered with a degenerated type of columnar epithelium. These areas suggest the active glandular proliferation of some types of hyperthyroidism and seem sufficient to class this tumor as a nodular toxic form. These areas, however, are so coarse and degenerated that they probably did not produce much thyroid secre-

tion. The walls of the blood vessels are unusually thick. Over a portion of the tumor, flattened out squamous epithelium of the tongue is seen.

Microscopic Diagnosis.—Lingual thyroid, nodular toxic type. J. M. Wainwright, M. D.

MEDICAL ARTS BUILDING.

BIBLIOGRAPHY.

1. Piersol, George A.: Human Anatomy. Phila., J. B. Lippincott Co., 1907; p. 1829.

2. Lahey, Frank E.: Lingual Goiter. Surg., Gynec. & Obst., 36:395; 1923.

REFERENCES.

a. Ziegelman, Edward F.: Lingual Goiter. San Francisco, Archives of Otolaryngology, Oct., 1932; Vol. 16, No. 4; pp. 406-505.

b. New, Gordon: Congenital Obstruction of the Larynx and Pharynx. J. A. M. A., 81:363 (Aug. 4), 1923.

c. Mayo, Charles H.: Diseases of the Thyroid; Collected Papers of the Mayo Clinic. Saunders Co., Phila., 1911; Vol. 3, p. 454.

d. Grace, Roderick V.; Weeks, Carnes: Lingual Goiter. New York, Annals of Surgery, Dec., 1932; Vol. XCVL., No. 6, pp. 973-978.

e. Armstrong, Harold G.: Thyroglossal Cysts and Fistula. St. Michael's Hospital M. Bull., 3:90, 1923.

f. Harvey, F.: British Jour. Surg., Vol. XIII, pp. 746-748; 1925-1926.

XXII.

CHONDROMA OF THE LARYNX: CASE REPORT.*

Harrington B. Graham, M. D.,

San Francisco.

REPORT OF TWO CASES.

Case 1.—Patient (about 60) as a youth had a fine singing voice. At 25 this quality in the voice was gradually lost and a roughness was noticed which, as years went on, was more pronounced when patient was physically exhausted or nervous. The first medical examination was made in January, 1933, when a smooth tumor was visible above the left vocal cord and which did not obscure the cord. A mass about the size of a small plum was palpable in the left side of the neck in the region of the thyroid cartilage; an X-ray showed a calcified enlargement of the left thyroid and cricoid cartilages. This has not changed in the year, and the function of the cords has remained as on first examination.

Case 2.—Patient has been under treatment for one year for ulcerative tumors of the larynx. There is a history of heavy drinking, negative Wassermann and negative T. B. examination. A biopsy specimen was reported as inflammatory tissue. I saw the case in November, 1933, when examination showed a loss of the left half of the rim of epiglottis. The surface was smooth as though there had been an ulceration which had healed in such a way as to leave the cartilage exposed. There was a tumor mass on the right side of the larynx which exhibited an ulcerative surface and obscured the vocal cord on that side. There was a large ulceration in the left pyriform fossa and a small isolated growth on the side of the pharyngeal wall. One lymph gland on the left side was enlarged but not tender. Blood Wassermann was again negative; blood count was normal and T. B. examination negative. The patient was kept on mixed treatment for two months with little effect on the lesions. A biopsy showed no evidence of carcinoma, T. B. or lues and was diagnosed as chondroma. The patient died in an insane asylum from a thrombosis of the cerebral vessels. No autopsy was permitted.

These two cases represent the extremes of a fascinating group which has been named chondroma but which includes evidently many clinical types. The picture one gets on studying the few recorded cases (77—May 19, 1932) reminds one of the more familiar carcinoma of the larynx rather than of the benign tumor it is supposed to be. One sees the small sessile growths which may

*Read at the Western Section meeting of the American Laryngological, Rhinological and Otological Society, Inc., January 12, 1934.

Case 1.

be easily removed per oram, never to return again, or the flat
infiltrating growths which gradually enlarge to fill the larynx,
killing by decreasing the breathing space. One sees, as in car-
cinoma, a smooth surface throughout the life history or a rap-
idly ulcerating surface. One sees a solid mass on the one hand
or a degenerative process, producing large cavities filled with a
mucoid secretion or a sago-like degenerated substance which may
be scooped out with a spoon. Some of the growths metastasize
or keep on invading adjacent tissue after operation, one recorded

Case 1.

case having produced death by invasion of the mediastinum and trachea to the bifurcation.

Although the size of the growth has in the majority of cases determined the end results, yet this is by no means universal, and one is tempted to suspect, as in my second case, that there may be a toxic element present which is having a marked influence on the status of the patient.

Irwin Moore has followed Virchow in his classification of these growths, dividing them into—

First—Ecchondromata arising from the deeper layer of the perichondrium and consisting of cartilage of the same structure as the cartilage of the larynx.

Second—Enchondromata or cartilaginous growths relatively benign, developing outside of the cartilage.

This classification was developed by Virchow for other parts of the body, but when applied to the larynx is extremely confusing

Case 2.
The cartilage appearing directly under the epidermal layer.

and probably misleading, as the end results of all the tumors may be myxochondroma, chondrosteoma and malignancy.

Like carminoma, they appear at all ages, but the more frequent period is between 40 and 60. The length of time that the tumor has been present is unknown as the patients seldom seek relief until the symptoms are pronounced, and this may be years after the inception of the growth.

The symptoms of the tumor are all dependent on the size and position, hoarseness, dyspnea, cough aphonia and dysphagia being present, according to whether the tumor is above or below the glottis and dependent in their character in large part on its size.

The diagnosis may be very easy or extremely difficult. In most well-developed cases the X-ray will show a density dependent to

a great extent on the calcification but also pronounced when no calcification is present. The tumor may be apparent by external inspection and palpation or by laryngoscopic examination, when it will appear pale in color, noninflammatory, as a rule smooth and hard on palpation with a sound. However, when ulceration and degeneration have occurred, as in my second case, it becomes a matter of grave importance to get a microscopic diagnosis.

OPERATIVE PROCEDURES.

The only treatment so far instituted has been operative, save one case recorded by Ingals, handled by application of chromic acid. This case is said to have decreased one-half in size, but the end result was not reported.

Seven cases were operated per oram, all successfully, but it is apparent that these must be small sessile tumors. A few were removed through laryngofissure, the tumor being either removed intact from the inside of the larynx or, in case it formed part of the wall, the cartilage was dissected from the mucous membrane. New, in one case, left a shell to form part of the new larynx; seven months later there was no return and the voice was normal.

In one case the opening in the larynx was carried only as high as the vocal cords, the tumor being removed with preservation of the voice.

A few had broken down and were removed by a spoon curette.

In general, the results have not been successful, many of the cases dying of pneumonia directly succeeding operation or of recurrences later on.

The bibliography of this subject has been thorouglhy covered by Irwin Moore, Journal of Laryngology and Otology, 40—1925; Clerf, Archives of Otolaryngology, 10:241, September, 1929, and Figi, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 41, No. 2.

490 POST STREET.

XXIII.

PAROTID GLAND TISSUE IN THE TONSILLAR FOSSA.

### Ernest M. Seydell, M. D.,

Wichita.

Name, Billie S., age 5 years. This patient was referred to me because of recurring attacks of tonsillitis, frequent head colds and enlargement of the cervical lymph glands. He was also a mouth breather. His physical examination was negative. The tonsils were hypertrophied and cryptic. The anterior pillars were injected and the cervical glands enlarged. His adenoid was hypertrophied.

The patient's tonsils and adenoid were removed under gas-ether anesthesia. After their removal, I noticed a small growth in the region of the lower pole of the right tonsil. My first impression was that I failed to remove completely all of the tonsil tissue. After the excision of a portion of this tissue, I realized that it bore no relation to the tonsil. It was yellowish in color and had a soft consistency. A careful examination of the tonsil fossa revealed that it contained more of the same tissue which extended outward and downward into the neck. No attempt was made to remove it.

A microscopic examination of the mass removed showed that it was parotid gland tissue.

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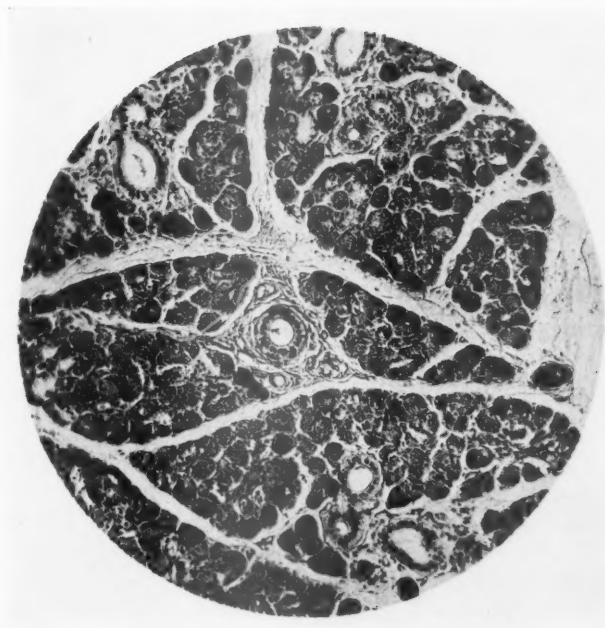
Parotid gland tissue.

XXIV.

UNILATERAL ATRESIA OF THE AUDITORY CANAL WITHOUT EXTERNAL DEFORMITY: A CASE.

COLBY HALL, M. D.,

LOS ANGELES.

There are three distinct reasons for the following report and discussion, namely, (1) the apparent rarity of this type of case, (2) the unusual and interesting history of this particular case, and (3) the embryologic explanation of the phenomenon presented.

Dean and Gittins,[1] in 1917, gave a very thorough report of the cases up to that time. There was one case in their investigation which showed a unilateral atresia without external deformity. The case reported by Dean and Gittins at this time was bilateral, unaccompanied by abnormal auricles, and was operated upon successfully.

Colver,[3] in 1932, reported a case of bilateral atresia of the external auditory meatus without deformity of the concha.

Fraser,[2] in 1931, reported fifteen cases coming to the Royal Infirmary of Edinburgh, all of them showing some malformation of the auricle. In addition, he made a very extensive review of the case records in the literature, and if any of these had atresia alone they were not mentioned. I am unable to state whether any of the fourteen cases that Beck reported a number of years ago were free of conchal deformity.

Fraser[2] says, "Congenital closure of the external meatus is, as a rule, associated with marked malformation of the auricle." Alexander[3] says, "The normal auditory meatus also fails to develop in these cases, and the skin at this place is either smooth or forms a short blind fossula from a few millimeters to one centimeter long. This deformity is usually associated with an irregularity of the concha, which may be entirely missing or replaced by several small shapeless cartilages, etc."

Thus it appears that meatal atresia unassociated with deformity of the auricle is rather a rare occurrence.

CASE REPORT.

M. K., age 47, female, white, married. Patient consulted me on September 7, 1933, because of some pain in the right ear following a mountain trip. Having had impaired hearing in the left ear since childhood she was naturally concerned about her only well-functioning ear. I had known this patient for four years, but this was the first time that she had consulted me professionally. During these four years she had repeatedly reminded me of the fact that her left ear was a "game one" and that she could hear very little with it. The patient had been told that during scarlet fever, at the age of 9, she had had an otitis media. Since then the ear had discharged from time to time, and one specialist had treated it for a period, and she was left with the impression that she had an "old scarlet fever ear."

Past history included the removal of a bifid uterus in 1923.

Examination: Right ear—Canal and drum normal. Left ear—Imagine my surprise to find a complete atresia just within the canal, perfectly evident by merely pulling the auricle upwards and backwards and without the use of a speculum. The blind end was lined with normal skin. The auricle was absolutely normal with no deformity of any type.

Nose—Very narrow spaces, which were too narrow for the passage of a eustachian catheter. Septum straight. Normal eustachian orifices were seen postnasally.

Tonsils—Moderate size, red, and ragged.

Hearing—Schwabach increased on the left. Rinne reversed on the left. Weber lateralized to the left. Hearing on the right was normal.

Audiometer:

	R.	L.
64	95%	0
128	85%	35%
256	97%	58%
512	100%	50%
1042	85%	52%
2048	85%	53%
4096	96%	49%
8192	87%	69%

The hearing is recorded in percentages of normal. The audiometric examination was done at the University of California, at Los Angeles, by Dr. Lawrence Gahagan of the Department of Psychology.

Turning:

To the right 10 times in 20 seconds—after nystagmus 24 seconds.
To the left 10 times in 20 seconds—after nystagmus 24 seconds.

Radiologic examination by Dr. Rolla G. Karshner.

"Stereoroentgenograms of the mastoids show a normal, well excavated cellular right mastoid. On the left excavation is not so extensive; the cells are smaller and the sinus groove is more distinct. The density of the entire mastoid and middle ear region is denser than it is on the right. The mastoid process is not cellular. Both the internal and external audi-

tory meatus can be distinguished on both the left and right. The right
external auditory meatus is more radiable than the left, but the difference
in radiability is not great enough to assume a bony plate obstructing the
left. The anterior canal wall is present on the left, and the distance be-
tween it and the mandibular condyle is equal to the similar distance on
the right. The only change demonstrable on the roentgenograms between
the left and right mastoid is the so-called infantile type of mastoid on the
left."

DISCUSSION.

As is clearly shown above, there are several interesting clinical,
as well as embryologic features in this case.

Of interest, of course, is the fact that until this examination
the patient believed that she had an O. M. P. C. following scar-
latina. The canal presented an absolute atresia, therefore the dis-
charge was most certainly ceruminous; the history excludes any
furunculosis, the occurrence of which might prove puzzling to one
not aware of the congenital defect. The question arises as to
whether the patient actually had a middle ear infection in child-
hood. The X-ray shows an infantile mastoid on the left, but we
cannot be certain that this resulted from an infection in childhood
or is an actual underdevelopment embryologically.

Dr. Karshner's report eliminates the possibility of the latero-
hyale having formed the bony atresia referred to by both Fraser[2]
and Richards.[4]

The history of the bifid uterus is of embryologic interest.

Normal turning reactions denote a normal vestibular component
of the inner ear. The functional hearing tests show an intact
cochlear component with a definite conductive type of deafness.
This intact and functioning internal ear merely emphasizes the
point brought out by both Richards and Alexander that this part
of the ear has no embryologic connection with any external or
middle ear structures and is usually normally developed in cases
of congenital atresia of the external auditory canal.

To explain the atresia we must refer to Richards'[1] recent work.
The expanded lateral end of the first gill cleft becomes the
middle ear and the narrowed medial portion becomes the eusta-
chian tube. Opposite the first gill cleft there forms an ectodermal
invagiation from which a row of ectodermal cells extends to the

middle ear. There is a canalization of this row from within out-wards, separating it into two layers, the inner layer forming the ectodermal layer of the definitive drum membrane. This canali-zation continues outward to meet the ectodermal invagination and form the external canal. Lyman Richards in one paragraph ex-plains fully the cause of the occlusion in this case.

"The absence of a patent auditory meatus rests fundamentally on the disturbance of the above described process of canalization of the ectodermal cord which extends inward from the depres-sion representing the external gill cleft. If the canalization fails to take place or if, having begun, it fails to extend to completion and thus make a connection with the external world by joining the gill cleft depression, then there will be a soft tissue occlusion of the canal which will be permanent."

Embryologically, the auricle and the meatus develop separately, but the deformity of one is usually associated with deformity of the other, as noted above, and the occurrence of a unilateral occlu-sion with two perfectly normal and uniform auricles is, I believe, quite uncommon.

Obviously no surgical procedure is indicated because of the presence of one normal functioning ear.

1136 W. Sixth Street.

REFERENCES.

1. Dean, L. W.: Trans. Amer. Laryn., Rhinol. and Otol. Soc., 1917.
2. Fraser, J. S.: Archives Otolaryngology, 13:1-28, January, 1931.
3. Colver, B. N.: Trans. Amer. Laryn., Rhinol. and Otol. Soc., 1932.
4. Richards, Lyman: ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1933.

POSTGRADUATE COURSE IN OTOLARYNGOLOGY: WASHINGTON UNIVERSITY SCHOOL OF MEDICINE, ST. LOUIS.

The Washington University School of Medicine offers annually a four weeks' intensive postgraduate course in otolaryngology. The object of the cause is to give otolaryngologists an opportunity of reviewing pathology, clinical diagnosis and operative work. Particular attention will be given to the basic sciences which today are responsible for the rapid strides that are being made in otolaryngology. The anatomy, the physics, the physiology, the embryology, the bacteriology and the cytology of the ear, nose and throat will be studied. The influence of nutrition and atmospheric conditions upon the ears and the nasal sinuses will be considered.

The course will be limited to qualified otolaryngologists. Those desiring to enter the practice of otolaryngology as a specialty should pursue at least one year of graduate work in otolaryngology before applying for admission to this course.

The major portion of the work will be given in the Oscar Johnson Institute, the Dispensary of McMillan Hospital, St. Louis Children's Hospital, Barnes Hospital, Ridge Farm, and Central Institute for the Deaf. Additional instruction will be given in St. Louis City Hospital and in the Edward Mallinckrodt Institute of Radiology.

The course is so arranged that the physician has his entire time occupied in intensive work during the month. The lectures, the demonstrations, the clinical conferences, and the round table discussions will be conducted before the whole group. When it is advisable to divide the class for laboratory demonstrations, bedside instruction, work in the dispensary, or for the demonstrations of operative work, such division will be made. The number of students is limited to twenty.

FACULTY.

OTOLARYNGOLOGY.

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C. C. Bunch, Ph. D., Professor of Applied Physics in Otolaryngology.
H. W. Lyman, M. D., Assistant Professor of Clinical Otolaryngology
M. F. Arbuckle, M. D., Assistant Professor of Clinical Otolaryngology
Arthur W. Proetz, M. D., Assistant Professor of Clinical Otolaryngology

Arthur M. Alden, M. D., Assistant Professor of Clinical Otolaryngology
I. D. Kelley, Jr., M. D., Assistant Professor of Clinical Otolaryngology
French K. Hansel, M. D., Assistant Professor of Clinical Otolaryngology
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gology
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Harry N. Glick, M. D., Assistant in Clinical Otolaryngology
Allen Potter, M. D., Assistant in Clinical Otolaryngology
Wm. F. Wenner, M. D., Instructor in Experimental Physiology in Oto-
laryngology
Dorothy Wolff, Ph. D., Instructor in Anatomy in Otolaryngology
Rossleene A. Hetler, Ph. D., Instructor in Applied Nutrition in Otolaryn-
gology

SPECIALTIES.

W. McKim Marriott, M. D., Professor of Pediatrics and Dean
Evarts A. Graham, M. D., Professor of Surgery
David P. Barr, M. D., Professor of Medicine
Max A. Goldstein, M. D., Director Central Institute for the Deaf
J. J. Bronfenbrenner, Ph. D., Professor of Bacteriology
Lawrence Post, M. D., Professor of Ophthalmology
Vilray P. Blair, M. D., Professor of Clinical Surgery
Ernest Sachs, M. D., Professor of Clinical Neurologic Surgery
Harry L. Alexander, M. D., Associate Professor of Medicine
J. J. Singer, M. D., Associate Professor of Clinical Medicine
I. Y. Olch, M. D., Assistant Professor of Surgery
J. F. Bredeck, M. D., Instructor in Clinical Medicine

The course begins on Monday, June 18, 1934. For further
information address Department of Otolaryngology, School of
Medicine, Washington University, St. Louis.

NOTICE.

During 1934, the American Board of Otolaryngology will hold
an examination in Cleveland, Ohio, June 11, 1934, during the
meeting of the American Medical Association; in Butte, Montana,
July 16, 1934, at the meeting of the Pacific Coast Oto-Ophthal-
mological Society, and in Chicago, September 8th, preceding the
meeting of the American Academy of Ophthalmology and Oto-
laryngology.

Applicants for certificate should address the Secretary, Dr. W.
P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska, for
application blanks.

Abstracts of Current Articles.

NOSE.

Hyperesthetic Rhinitis.

Fox, Noah, and Fabricant, Noah D. (Chicago), Arch. Otolaryng., 18:181 (August), 1933.

Recognizing the confused nomenclature of this condition and finding only 1 per cent of their patients with boggy membranes to have nasal allergy, the authors suggest that the terms hyperesthesia, allergica, vasomotoria, suppurativa and neuralgica be added to the name of the underlying hyperplastic sinus condition (sinusitis hyperplastica) to classify it.

Pale color of the nasal mucous membranes due to waterlogging of the tissues occurs in infections and allergy, microscopically the only difference of cellular elements in allergy and chronic inflammation is the presence of eosinophiles in the former.

Patients with a nasal sensibility should be desensitized, but where the sinuses have hyperplastic disease, ventilation and drainage are indicated, as radical as necessary.   TOBEY.

Diagnosis and Treatment of Primary Malignant Neoplasms of the Maxillary Sinus.

Houser, Karl Musser (Philadelphia), Arch. Otolaryng., 18:643 (Nov.), 1933.

This paper deals with the diagnosis and treatment and end results in twenty-one cases of primary malignant tumor of the maxillary sinus and is discussed by Drs. Sewall, Guttman and Havens.

Early diagnosis is urged by opening the sinus for inspection and biopsy. The author feels that surgical procedures are necessary for adequate drainage but are chiefly useful in exposing the lesion for direct irradiation.

Roentgen and radium therapy in adequate dosage offer the most hope of cure.   TOBEY.

PHARYNX.

Sarcomatous Tubular Hemangiomesothelioma at Base of Tongue, of Probable Thyroidal Origin (Emoangiomesotelioma tubulare sarcomatose della base della lingua di probabile origine tiroidea).
Mesolella, V. (Naples), Arch. Ital. di Otol., Rino. E Laryng., 44:477 (August), 1933.

The writer has studied, in a young woman, aged 18, a tumor at the base of the tongue, which at the histologic examination was revealed to be a tubular sarcomatous hemangiomesothelioma (six microphotographs). He does not exclude the possibility that such neoplasms could develop from an aberrant goiter of the tongue, as its base was attached at the location of the foramen cecum, where thyroid residue may be found; but this supposition is not confirmed by the histologic findings, even though in various sections structures similar to the thyroid gland are seen. He describes the embryology of the thyroglossal duct, the symptomatology of the lingual goiter and the structure and classification of the endothelioma.   SCIARRETTA.

Etiopathogenic Consideration of Peritonsillar Phlegmon (Considerazioni etiopatogenetiche sul flemmone pertonsillare).
Giuffrida, E. (Catania,) Arch. Ital. di Otol., Rino. E Laryng., 44:449 (August), 1933.

The author, in this long and interesting article, reviews the literature extensively in respect to the bacteria in this lesion, the pathologic variations as to age, seasonal predomination and frequency of kidney involvement. He further draws the following conclusions from 121 cases of peritonsillar phlegmon studied in his clinics during the years of 1931 and 1932.

Types of bacteria: Pure diplococcus (gram positive), identified as pneumococcus, 25 per cent; pure streptococcus, 23 per cent; pure staphylococcus, 1 per cent; mixed diplococcus and bacilli (mostly coli B.), 33 per cent; mixed diplococcus and streptococcus, 11 per cent; mixed diplococcus and staphylococcus, 4 per cent; fusospirilla of Vincent, 2.5 per cent.

In adults the phlegmon invariably forms an abscess, while in children, if treated with moist heat, it will absorb. In certain types of individuals, especially uremics, when the phlegmon is incised, you obtain blood instead of pus. The septic type is produced by a pure streptococcus infection, which brings about systemic com-

plications (pyemia, metastasis, endocranial complications and hemorrhages by erosion of the blood vessel walls).

The epidemic manifestation of this malady, occurring during certain periods of the year (December and January, and June and July) seems to be caused by the thermic and climatic variations and not by a certain type of bacteria.

Parenchymatous nephritis is found only in individuals predisposed to it: in all, 2 per cent. Transitory albuminuria was observed in 37 per cent of the cases; of these, 20 per cent were infected by streptococcus and 13 per cent by pneumococcus.

<div style="text-align:right">SCIARRETTA.</div>

LARYNX.

Cysts of the Larynx.

Myerson, Mervin C. (New York). Arch. Otolaryng., 18:281 (Sept.), 1933.

Cysts of the larynx are of four types of retension, embryonal or congenital lymph or blood and traumatic or implantation. All these types are described and three infant cases are reported in detail, where cysts had been present from birth. Cysts in infants are very serious and probably the best treatment is cautery incision which permits drainage until the patient is able to stand removal. Symptomatology, diagnosis and prognosis are discussed.

<div style="text-align:right">TOBEY.</div>

Innervation of the Larynx—Experimental Paralysis of the Laryngeal Nerve.

Lemere, Frederick (Denver). Arch. Otolaryng., 18:413 (October), 1933.

The author agrees with Onodi rather than Luschka as to innervation, because no one has ever produced contraction of any of the laryngeal muscles except the cricothyroid by stimulation of the superior laryngeal nerve.

The action of each laryngeal muscle in the dog was studied by isolating it on an otherwise stripped laryngeal skeleton and stimulating it electrically. For all practical purposes the larynges of man and dog were found identical. The effect of sectioning the various laryngeal nerves on the glottic picture were studied and reported.

The author feels that after section of both recurrent nerves the overacting cricothyroids soon atrophy, due to lack of apposition by the thyro-arytenoids, and thereby more breathing space is obtained, after a time.

<div style="text-align:right">TOBEY.</div>

EAR.

Severe Deafness in Adults.

Shambaugh, Geo. E.; Wallner, L. J.; Greene, L. D.; Shambaugh, Geo. E., Jr. (Chicago), Arch. Otolaryng., 18:430 (October), 1933.

The authors studied 165 adults suffering from handicap deafness and most of these were members of the Chicago and Washington Leagues for the Hard of Hearing. The remaining ones were from their private practices. A tabulated summary of sixty-five cases is given, and of these thirty deaf adults were afflicted with primary nerve deafness; five due to toxic neuritis from an acute infectious disease, two to a congenital defect, two due to a noisy occupation and seven due to senile changes. Syphilis was a likely cause in one case. In twelve cases the cause was unknown.

Of the 165 standard cases twenty patients had tubotympanic middle ear deafness and 115 were diagnosed as otosclerosis.

Shambaugh and his workers believe that only 12 per cent of severe adult deafness is due to middle ear disease of childhood and are preventable, and that 70 per cent of severe adult deafness is due to otosclerosis. TOBEY.

Surgical Importance of the Mastoid Vein in Infected Lateral Sinus Thrombosis.

Ziegelman, Edward F. (San Francisco), Arch. Otolaryng., 18:298 (September), 1933.

The author has no case reports but has examined thirty mastoid emissary veins, postmortem, and feels that in any cases suspected of an infected lateral sinus, examination of the mastoid emissary vein should be carried out. If found to be thrombotic, it should be ligated to avoid systemic invasion by a portal other than the internal jugular. In 25 per cent of specimens examined, the mastoid vein was absent. TOBEY.

Objective Tinnitus Aurium With Report of Four Cases.

Iglauer, Samuel (Cincinnati), Arch. Otolaryng., 18:145 (Aug.), 1933.

In this article only the vascular type of tinnitus is considered and mere mention made of the type in which the eustachian tube muscles are causative. Four cases are reported, in three of which the noise seemed to originate in the jugular bulb, as rotation of the head in certain directions could produce or stop the tinnitus. Ligation of the internal jugular vein is proposed for treatment in well-selected cases. TOBEY.

A New Application of the Weber Test (Una nuova applicazione della prova del Weber (Preliminary Report).

Up to date the Weber test has been used to locate the diseased ear or the one most affected and also to determine which portion is involved. In addition to the above uses, the author employs the Weber test to determine the degree of the involvement of the middle ear and the functional difference of the two sides.

Technic: In a lateralized Weber the fork is moved gradually from the center of the cortex towards the mastoid tip of the normal or better ear until the sound is heard equally in both ears: therefore it follows that the further this point is from the center the greater is the functional difference, as compared with the other ear, and also the greater is the degree of middle ear involvement. If, at a later date, this test is repeated you may also determine the amount of improvement, if any.

SCIARRETTA.

MISCELLANEOUS.

Involvement of the Esophagus in Acute and in Chronic Infection.

Dr. Mosher discusses the postmortem findings of 100 autopsies, both macroscopically and microscopically. He is of the opinion that cardiospasm (ventriculosis) is due to the formation of fibrous tissue in the subepithelial layer or in the circular muscular layer of the lower end of the esophagus. This is preceded by infiltration of polymorphonuclear and small round cells. He also found superficial infections, infected follicles, flaking of the epithelium, replacement of muscular layer with fibrous tissue, one broken down gumma, etc.

He concludes that the modes or routes of infection are by direct contact through the blood and lymph stream, bronchi, pleura, peritoneum, and especially from liver and gall bladder disease. "The esophagus, to recapitulate, can be infected from within and is often infected in both acute and chronic diseases." He also discusses his methods of treating ventriculosis.   TOBEY.

A New Diagnostic Sign for Early Diagnosis of Esophageal Cancer (Arslan's sign) (Un nuovo segno semiologico per la diagnosi precoce di cancro esofageo).

Rubaltelli, E. (Padova), Boll. del'e Malatt. Orecch. Gola, Naso, 50.282 (September), 1932.

The sign consists of an initial and superficial infiltration at the arytenoid process of the same side or both processes if the lesion involves both esophageal walls. The tissue covering the arytenoid process or processes presents an increase in size with hyperemia, edema with infiltration and uniform distention covered with secretion. This condition can be easily differentiated from the well-known pretubercular infiltration of the larynx which is so familiar to the laryngologist. This sign is constant when the neoplasm involves the upper third of the esophagus. It appears very early and long before the patient complains of any symptoms of obstruction or function.   SCIARRETTA.

Pulmonary Symptoms Due to Esophageal Disease.

Jackson, Chevalier, and Jackson, Chevalier L. (Philadelphia), Arch. Otolaryng., 18:731 (December), 1933.

The authors discuss the pathologic conditions of the esophagus which cause pulmonary symptoms; their early diagnosis, prophylaxis and treatment. They list the nine ways and means by which pulmonary symptoms are produced by pathologic conditions in the esophagus and hypopharynx. Then they take each one separately and discuss it. They are as follows:

1. Inspiration of infected food, blood or secretions (oral, pharyngeal, nasal) which overflow into the larynx because they cannot get down a stenosed esophagus.

2. Direct extension upward and over the laryngeal rim by pathologic processes originating in the esophageal or hypopharyngeal wall.

3. Direct extension through the esophagotracheal party wall or through the wall of a bronchus, usually the left main bronchus.

4. Direct extension of esophageal disease to the pleura or through the pleura into the parenchyma of the lung.

5. Direct extension of an esophageal lesion to the mediastinum, thence to the lung.

6. Extension of esophageal disease by way of the blood vessels or lymph channels.

7. Compressive stenosis of the trachea or bronchus without pathologic involvement of the tracheobronchial wall, by bulky esophageal lesions or esophageally lodged, large foreign bodies.

8. Laryngeal paralysis by pressure or involvement of the recurrent nerve by a lesion, such as a carcinoma.

9. Reflex symptoms, especially cough, excited by disease limited to the esophagus.

In their conclusions they emphasize the importance of early diagnosis and treatment.   TOBEY.

"Should Fusospirochetal Infections Be Treated With Arsenicals?"— Report of Cases.

Smith, David T. (Durham, S. C.), Arch. Otolaryng., 18:760 (Dec.), 1933.

The author discusses the clinical and experimental evidence in favor of fusospirochetal organisms as being the primary factor in causing lesions of the respiratory tract in which they are found. He is of the opinion that this evidence is sufficient to warrant it as the etiologic factor.

The treatment of Vincent's angina (both in the young and the old patients), chronic fusospirochetal infections of the larynx, chronic trench mouth, Vincent's infections in avitaminosis, certain postoperative pulmonary infections and pulmonary abscess (when the fusospirochetal organisms are found) should have arsenical therapy, usually intravenously but intramuscularly when necessary. He usually uses one to four doses and prefers not to use large doses but at more frequent intervals. He illustrates each condition with a case history.   TOBEY.

Anemia in General Practice.

L. J. Witts (London), The British Medical Journal, June 24, 1933.

Anemia is frequently overlooked and it is often difficult to be certain of the presence of anemia from clinical inspection. The white face of the sedentary worker usually hides a normal blood, while the invalid who has been lying in the sunshine may have a sunburnt complexion with a marked anemia. The conjunctiva and the palate best reflect the state of the blood. Treatment should not be given for anemia until the diagnosis has been confirmed by the hemoglobinometer. Symptoms of anemia rarely appear except in certain cases of pernicious anemia until the hemoglobin

has fallen below 75 per cent. The nature of the anemia should·
be determined and a complete blood count performed.

Symptomatic anemias are far more common than primary dis-
eases of the blood-forming organs, and they rarely respond to
treatment until the underlying disease has been corrected. A case
of anemia requires a detailed history and complete investigation.
In discussing the causes of secondary anemia the writer empha-
sizes the importance of chronic nephritis and deprecates the attri-
bution of anemia to intestinal toxemia. Malignant disease should
be considered in every case of obscure anemia. While it is impor-
tant to exclude gross organic disease in a case of anemia it is
generally important not to attribute it to trivial and unreasonable
causes. The writer has rarely seen amenia much improved by
the eradication of foci of sepsis or the treatment of a positive
Wassermann reaction and such findings are more often co-
incidences than etiologic factors.

The primary anemias are classified into four main groups.
Leukemia and Hodgkin's disease show great symptomatic im-
provement with X-rays or arsenic, though life is probably but little
prolonged. The second group, hepatorenal diseases, treated with
liver and iron may show transient improvement, but the only sat-
isfactory treatment is splenectomy. In hemorrhagic diseases, ex-
cept the group with reduction in the number of blood platelets
which is benefited by splenectomy, the treatment is unsatisfactory.
The anhemopoietic anemia is the fourth and important group,
where the nutrition of the bone marrow is impaired and its ability
to form blood cells correspondingly reduced. It may be the results
of defects in diet, digestion, or in the internal organism, espe-
cially the endocrine glands. Many conditions give rise to anemia
in this way, but the two most important are iron deficiency and
pernicious anemia. The former is found frequently in infants and
in women of reproductive age and the treatment is iron in large
doses. Anemia in infants leads to an increased susceptibility to
infection. Colds, otorrhea and bronchitis are twice as common
in untreated babies as in those in which the anemia is treated with
iron.

The unsatisfactory results of pernicious anemia are due to
inadequate treatment with whole liver, extracts of liver or desic-

cated stomach. It is impossible to give an overdose of effective substance and the treatment need in no way interfere with the employment of a normal diet and active life. Cases should be kept under regular supervision to see that this hemoglobin is well maintained. There is no more reason for patients with pernicious anemia to succumb to subacute combined degeneration than for diabetics to die in coma. Both complications are preventable by careful treatment.   GOLDSMITH.

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